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BOVINE TUBERCULOSIS IN MAN.[1]

By H. V. D. BARET, B.A., M.B. (Sydney),
Director of the Tuberculosis Division, Department of Public Health, New South Wales.

IT is with a great deal of diffidence that I approach the subject of the relation of tuberculosis in cows to the presence of bovine tuberculosis in human beings. The data available in New South Wales are so insufficient that any conclusions must be only tentative. Yet I think that inadequate as are the figures, fairly definite impressions may be formed.

We have to consider (i) the presence of tuberculosis in cattle, (ii) the presence of bovine tubercle bacilli in the media of infection, (iii) the presence of bovine tuberculosis in human beings.

THE INCIDENCE OF TUBERCULOSIS IN CATTLE.

The incidence of tuberculosis in cattle may be judged by abattoir *post mortem* examinations, the result of tuberculin tests, the examination of dairy cattle on the hoof and bacteriological examination of milk.

Abattoir Evidence.

The abattoir figures are given first.

Sydney Abattoirs.

For fifteen months ended June, 1917, the number of cattle slaughtered was 138,335; of this number 1·3% were totally condemned for tuberculosis and 5·2% partially condemned, making a total of 6·5%.

For the twelve months ended December, 1924, the number of cattle slaughtered was 236,790; of these 0·6% was totally condemned for tuberculosis and 1·0% partially condemned, making a total of 1·6%.

For the twelve months ended June, 1927, the number of cattle slaughtered was 355,595; of these 0·6% was totally condemned for tuberculosis and 1·0% partially condemned, making a total of 1·6%. The number of calves killed was 108,121; one calf only was condemned.

Newcastle Abattoirs.

For the twelve months ended December, 1924, the number of cattle slaughtered was 34,756; of these 0·7% was totally condemned for tuberculosis and 4·7% were partially condemned, making a total of 5·4%.

For the twelve months ended June 30, 1927, the number of cattle slaughtered was 35,265; of these 1·1% were totally condemned for tuberculosis and 3·5% partially condemned, making a total of 4·6%. The number of calves killed was 3,807; one calf only was condemned.

These figures may be compared with those for the cities of London and Edinburgh.

There is a good deal of clinical evidence as to the great prevalence of tuberculosis in New South Wales twenty or thirty years ago, but a great decline in the disease has been noted clinically also. This observation is supported by the abattoir evidence.

[1] Read at a meeting of the New South Wales Branch of the British Medical Association and the Veterinary Association of New South Wales on November 28, 1929.

TABLE I.

City of London, 1924.

Animal.	Number Slaughtered.	Percentage Tuberculous.
Bulls	160	4·40
Calves	34,853	0·04
Cows	284	28·00
Heifers	64	2·00
Oxen	4,697	3·10

City of Edinburgh.

Animal	1926.		1927.	
	Number Slaughtered	Percentage Tuberculous	Number Slaughtered	Percentage Tuberculous
Bulls ..	476	15·33	603	12·62
Calves .	4,818	0·24	4,936	0·67
Cows ..	3,479	46·50	3,687	42·99
Heifers .	747	7·89	953	5·67
Oxen ..	27,187	3·88	28,045	3·73

Results of Tuberculin Test.

No large body of statistics is available, but the following figures I owe to Mr. Bloomfield, Chief Dairy Inspector.

A consignment of 1,730 dairy heifers purchased for the Western Australian Government and obtained from the south and north coast, was tested and 3·1% reacted.

Mr. Symons, Chief Veterinary Surgeon, tested 1,163 head of cattle and 3·1% reacted.

The present Chief Veterinary Surgeon tested 1,061 dairy cows and 5·4% reacted.

A dairy herd of thirty-four contained two reactors.

Another herd of thirty-six contained three reactors.

A herd of 101 cattle contained five reactors.

These results are tabulated as follows.

TABLE II.

Number of Cattle.	Percentage of Reactors.
1,730	3·1
1,163	3·1
1,061	5·4
34	5·9
36	8·3
101	5·0
4,125	—

It is difficult to know how to apply these figures to the general cow population without knowing the exact circumstances surrounding each testing, so as to eliminate any question of selection. The factor of age, too, is not brought out.

In England it is generally said that 33% of the dairy herds are tuberculous in some form or other.(1)

Evidence from Examination of Cattle on the Hoof.

In different areas of England and Scotland the inspection of cattle in New South Wales.

TABLE III.

	1915	1923	1926
Number examined	169,920	176,830	428,716
Number condemned	0·3%	0·18%	0·08%

In two areas in England, Cumberland and Gloucester, in 1926 to 1928, the number examined was 163,343 and the proportion condemned was 0·46%.

In the next table are set out the results of the percentage varies from 0·15 to 1·77.

Evidence from the Bacteriological Examination of Milk.

The Bureau of Micro-biology has done some interrupted work in the investigation of tubercle bacilli in the Metropolitan milk supply. The work was originally commenced in 1921, but for various reasons has not been continuous. Out of a total of 151 samples of milk examined, there was evidence of tubercle bacilli in one and another was doubtful. This investigation has been continued by Dr. Morgan during 1927, 1928 and 1929.

In 1927 230 samples were examined, in 1928 212 and in 1929 75. No tubercle bacilli were found in any of the samples and no tuberculous lesions were produced in injected guinea-pigs.

Confirming these figures are those of the Dairy Farmers' Co-operative Milk Company, Limited; during the past three years 348 samples have been examined and on no occasion were tubercle bacilli found. Each of the samples tested was taken from the bulk supply drawn from various centres and would represent anything from 60 to 200 suppliers.

The extraordinarily small percentage of milk samples containing tubercle bacilli may be compared again with English experience, where variations have been found from 2% to 15% or even more.

The four heads that have been cursorily considered above, are consistent in their results with each other. They show that we are in a vastly better position as regards tuberculosis than is England and they show that the incidence of tuberculosis in cattle is rapidly falling.

As regards the absolute amount of tuberculosis in cows, we can estimate it at 4%, a rough average of the available tuberculin tests.

Professor J. D. Stewart in 1919 made an estimation of 8%.

Even if we accept this higher figure we must take into consideration the fact that in the majority of cows that react, only small lesions of tuberculosis are found which could not possibly have any influence in infecting milk. It may be said, of course, that reactors will harbour open lesions later on. On this point I should be glad to have the experience of the veterinary surgeons as to whether the course of the disease is downwards or, as in human beings, does the great majority of reactors conquer the disease.

The Incidence of Tuberculosis in Man.

The next point for consideration is the incidence of bovine tuberculosis in man. The only investigation of types of tubercle bacilli that I know of in Australia is that of W. J. Penfold. The following table is given by him.

He remarks that "the chief fact to be learned from this table is that no less than 26% of the infections of the first four years of life are bovine in origin and though the numbers are small, they clearly demonstrate that bovine tuberculosis is a very definite menace to Melbourne children."

A curious feature about the table is that after the age of four all the infections are of the human type.

Judging by the English figures available which show results widely discordant for different districts, it would be hazardous to apply the results of Dr. Penfold's investigation to Sydney.

As pulmonary tuberculosis is almost exclusively due to the human type of bacillus, the non-pulmonary types of tuberculosis may be considered.

The following table reveals the fall in death rate from pulmonary and other forms of tuberculous disease averaged over five-year periods.

TABLE V.

Five-year Period.	Death Rate per Million of Population.	
	Pulmonary Tuberculosis.	Other Tuberculous Diseases.
1891-1895	867·2	304·2
1896-1900	796·2	238·0
1901-1905	825·8	168·8
1906-1910	663·4	148·2
1911-1915	633·2	104·6
1916-1920	571·4	84·8
1921-1925	503·4	58·4

The actual number of deaths for the last quinquennial period are given in the next table.

The decline in non-pulmonary tuberculosis has not proceeded on lines parallel or proportional to the decline in pulmonary tuberculosis. Another factor is probably involved which is no doubt the decline in bovine tuberculosis.

TABLE IV.

Ages of patients from whom material was taken, arranged in groups of bovine and human infections.
(Melbourne Children's Hospital.)

Types of Bacilli.	Age Periods (Years).															All Ages.
	Unknown.	0–1	1–2	2–3	3–4	4–5	5–6	6–7	7–8	8–9	9–10	10–11	11–12	12–13	13–14	
Bovine ..	1	1	1	4	2	0	0	0	0	0	0	0	0	0	0	9
Human ..	0	7	5	8	2	2	2	3	0	2	2	2	0	2	2	39

TABLE VI.

Year.	Pulmonary Tuberculosis.	Non-Pulmonary Tuberculosis.
1921	1,129	149
1922	1,080	139
1923	1,114	130
1924	1,165	91
1925	1,023	127

In the following table the number of deaths from tuberculosis of children under five years of age is given.

TABLE VII.

Year.	Pulmonary Tuberculosis.	Non-Pulmonary Tuberculosis.	Total.
1908	16	85	101
1909	21	91	112
1910	10	96	106
1911	9	82	91
1912	8	81	89
1913	9	118	127
1914	8	82	90
1915	8	77	85
1916	17	67	84
1917	4	64	68
1918	11	60	71
1919	9	56	65
1920	9	58	67
1921	7	58	65
1922	7	46	53
1923	12	38	50
1924	8	33	41
1925	5	38	43
1926	6	47	53
1927	6	42	48

This table shows also a progressive improvement for both classes of tuberculosis. The improvement is greater than appears on account of the increased number of children.

A further table is added, showing deaths from meningitis and abdominal tuberculosis in children under five years.

TABLE VIII.

Year.	Meningitis.	Abdominal Tuberculosis.
1908	37	40
1909	48	37
1910	57	34
1911	42	30
1912	43	23
1913	76	26
1914	49	19
1915	48	13
1916	39	11
1917	37	12
1918	39	9
1919	15	5
1920	34	8
1921	38	5
1922	34	8
1923	29	7
1924	26	5
1925	29	2
1926	37	4
1927	28	5

Again the figures are better than appears on account of increased number of children. Their importance is that in English experience 20% of meningitic infections and 80% of abdominal tuberculosis are of bovine origin. If such proportion holds in New South Wales, then bovine infection must be at a minimum as shown by the greatly diminished number of deaths from abdominal tuberculosis. There is also a clear improvement, but not to such a great extent, in the meningitic infections where the human element of infection is still persistent.

Without giving the actual tables I may say that the figures for abdominal tuberculosis are much better for the ages from five to nine and still better for the ages from ten to fourteen.

The foregoing tables demonstrate a rapid decrease in non-pulmonary tuberculosis and in that division of it, abdominal tuberculosis, most closely associated with the bovine bacillus. Naturally we correlate it with the well recognized decrease in tuberculosis among cows, but we have no means at present of giving accurate statistics on the subject.

Yet I think a strong impression will be left that under present conditions there is no need for undue alarm in New South Wales concerning bovine tuberculosis in human beings.

That absence of undue alarm would, however, be no excuse for not attempting to stamp it out altogether. The first precaution is pasteurization. I do not know of any experiments that prove raw milk to be of greater value than pasteurized milk. The consensus of opinion seems to be that milk should be pasteurized at a temperature of 63° C. (145° F.) by a holding process for thirty minutes. However, as pasteurizing plants may fail either through the mechanical or through the human element, it will still be essential by thorough dairy inspection to continue to educate the dairyman in sanitary methods.

Graded milk is, I think, objectionable in that the poorer classes will inevitably buy the cheaper milk.

Should cows that give a tuberculin reaction, be eliminated from the herds? If a positive reactor were a positive actual danger the answer would be easy. But it is known that in the great majority of cows only minor lesions are revealed *post mortem* and the impression gathered from the results of examinations of milk and the consideration of the mortality in children is that the problem is not urgent, provided that pasteurization is performed.

The practical difficulty is that in an endeavour to save a few children in one direction, we shall lose many more by depriving them of a valuable food through the increase in the price of milk. I have, however, nothing to say against the idea of a voluntary accredited herds scheme. The idea should be encouraged. A system of rewards or punishment could be evolved. Compensation could, for instance, depend on whether tuberculosis in a cow was clinically detectable or not.

If in the course of this paper questions have been raised whose answers are not evident, then the primary purpose has been achieved, which is to excite discussion and thereby to clarify the points on which we need further data.

For much in this paper, especially where reference is made to English and Scottish statistics, I wish to express my acknowledgements to Dr. W. G. Savage whose book on "The Prevention of Human Tuberculosis of Bovine Origin"(2) is most stimulating.

REFERENCES.

(1) J. W. Brittlebank: "Clinical Diagnosis of Bovine Tuberculosis," *Veterinary Record*, May 12, 1928, page 371.
(2) W. G. Savage: "The Prevention of Human Tuberculosis of Bovine Origin," London, 1929.

TUBERCULOSIS AND MEAT INSPECTION.[1]

By J. DRABBLE, B.V.Sc. (Sydney),
Pathologist, State Abattoir, Homebush Bay, New South Wales.

TUBERCULOSIS affects all food-producing animals and is the disease most frequently met with by meat inspectors. It is fairly safe to say that most of the modern systems of meat inspection in the various countries of the world owe their existence largely to the fact that food animals are commonly affected with tuberculosis and to the possible danger of human beings contracting the disease by eating meat from affected animals.

Tuberculosis is common in cattle and pigs, but is rare in sheep. As a matter of fact no case of this disease in Australian sheep has yet been recorded and for that reason one almost seems justified in regarding the sheep of this continent as being immune to natural infection with tuberculosis.

Very few figures are available regarding the incidence of tuberculosis in food animals in New South Wales. Most of the official statistics obtainable are those relating to condemnations. Condemnations are quoted as total or partial. Total condemnation means condemnation of the whole carcass and all the organs. In official records partial condemnation means condemnation of one or more quarters or of the head including the tongue. Condemnations of organs such as the lungs, liver or intestines are not recorded and for this reason the official figures relating to condemnations for tuberculosis do not give any real information as to the incidence of the disease in food animals. Cases of tuberculosis in which the lesions are slight and confined to the bronchial, mediastinal or mesenteric lymph glands or to a small area of the lung, are quite commonly met with in both cattle and pigs.

During the past nine or ten years the incidence of the disease in cattle and pigs passing through the New South Wales State Abattoir has been fairly uniform and this uniformity has also been more or less evident in the percentages of condemnations for this disease during the same period. The incidence of tuberculosis in food animals does not necessarily run parallel with the percentages of condemnations for the disease. The modern tendency to slaughter beef cattle at an earlier age than was customary some years ago might conceivably have the effect of lowering the percentage of condemnations for tuberculosis in cattle while the incidence of the disease in these animals remained more or less stationary or even showed an increase.

[1] Read at a combined meeting of the New South Wales Branch of the British Medical Association and of the Veterinary Association of New South Wales on November 28, 1929.

The percentages of condemnations of cattle and pigs at the New South Wales State Abattoir for the years 1921 to 1928 inclusive are as set out in Table I.

TABLE I.

Showing Percentages of Animals Condemned in New South Wales State Abattoir.

Year.	Species.	Number Slaughtered.	Percentage of Animals Totally Condemned.	Percentage of Animals Partially Condemned.	Percentage Totals.
1921	Cattle	170,882	0·52	1·10	1·62
	Pigs	80,606	0·70	1·99	2·69
1922	Cattle	210,927	0·43	1·10	1·53
	Pigs	114,766	0·69	1·27	1·96
1923	Cattle	242,893	0·49	0·76	1·25
	Pigs	153,241	0·95	1·56	2·51
1924	Cattle	233,930	0·58	0·94	1·52
	Pigs	134,521	1·04	2·02	3·06
1925	Cattle	238,339	0·60	0·98	1·58
	Pigs	141,132	0·44	1·60	2·04
1926	Cattle	234,925	0·91	1·64	2·55
	Pigs	182,439	0·58	1·49	2·07
1927	Cattle	255,588	0·55	1·61	2·16
	Pigs	194,263	0·59	1·09	1·68
1928	Cattle	225,785	0·72	1·48	2·20
	Pigs	179,531	0·56	1·32	1·88

The progressive increase in the relative number of young beef cattle slaughtered at the State Abattoir in recent years would naturally lead one to expect that if any change took place at all in the percentage of condemnations for tuberculosis in cattle, it would be a decrease. It will be seen, however, that the percentage of 1·58 in 1925 increased to 2·55 in 1926 and remained at over 2·0 in 1927 and 1928. The most probable explanation of this is that the increase was due to an increased percentage of condemnations of dairy cows.

The figures for the years 1921 to 1928 inclusive show a definite and sustained improvement over those quoted by Dr. Baret for 1917. For some years prior to 1917 the percentage of total condemnations for tuberculosis in cattle was about 1·2 and that for partial condemnations was a little over 2·0. During the same period the percentage of total condemnations for tuberculosis in pigs was approximately 1·5 and that for partial condemnations ranged from 2·5 to about 4·5.

Dr. Baret's estimate of 5% as being the present incidence of tuberculosis in cows appears to me to be somewhat conservative, as I take it that he is referring solely to the dairy breeds. While this percentage may be correct for cows actually in dairies which are subject to regular inspections by experienced dairy inspectors, it would not be high enough to include also cows owned by various institutions, dealers, hotel-keepers, boarding-house keepers and private householders.

Professor Stewart's estimate of the incidence of tuberculosis in dairy cows in 1919 at 8% was not excessive for that period.

As a general rule the course of the disease in milch cows is very definitely progressive. This is due in a large measure to the great strain of heavy milk production on the animal's system and also to the rather too common practice of allowing dairy cows during the non-lactating period to fend for themselves in paddocks where feed is sometimes scarce. As a result of this the physical condition and powers of resistance of the animals are often lowered beyond safe limits and when calving time comes along, many cows are physically unfit to face once again the great task of milk production. Under such circumstances it is easy to understand why dairy cows are more susceptible to tuberculosis than are other cattle.

Very few people realize that the milk produced by a good dairy cow in one year contains over 55% more protein, 30% more non-nitrogenous nutrients (fat and carbohydrates) and about 19% more mineral matter than is contained in the entire body of a fat bullock weighing 540 kilograms (1,200 pounds). Over one half of the total mineral matter consists of lime and phosphoric acid.

This great output of lime indicates the need for a liberal supply of calcium in the food of dairy cows. When the supply is below her requirements, the dairy cow makes up the deficiency by withdrawing lime from the bones of her skeleton. Another very important point is that any deficiency in calcium assimilation definitely lowers the animal's powers of resistance to tuberculosis.

Although it is universally acknowledged that the majority of cases of bovine tuberculosis in human beings is due to the consumption of raw milk from tuberculous cows, the possibility of infection as a result of eating raw or partly cooked meat from tuberculous animals, especially where meat inspection and supervision are inefficient or non-existent, cannot be excluded. It is well known that meat, whether consumed in the form of roasts, grills or as sausages, is often eaten in a semi-raw condition and, although it may be urged that the voluntary muscles rarely contain tuberculous lesions or tubercle bacilli, one has to remember that the term "meat" includes not only the striated muscles, but also bones, fat, connective tissue, blood vessels, lymph vessels and even lymph glands.

Where supervision is lax or absent, meat, including the striated muscles, often becomes seriously contaminated with tubercle bacilli by means of knives, choppers or saws that have passed through tuberculous lesions. Under such circumstances, even if some attempt is made to wash the soiled implements before further use, it is highly probable that the washing is done in the tub of water which is used for washing the carcasses, and with the aid of a hessian wiper that is afterwards used to wipe carcasses of animals subsequently slaughtered.

Similarly a carcass is sometimes badly contaminated during the process of dressing through the accidental opening of a tuberculous abscess containing fluid pus. The contents of the abscess flow down the carcass and may soil one or both sides. After surveying the mess for a moment, the slaughterman, taking advantage of the absence of proper supervision and inspection, removes the abscess with his knife, dashes a bucket of water on the soiled portions of the carcass and then by means of a wiper endeavours to clear away any obvious pus that the water has failed to dislodge.

It is apparent that when such contaminated meat is eaten in a semi-raw condition, it is hardly less dangerous to the consumer than is the consumption of raw tuberculous milk. If it be contended that in such cases the contaminating bacilli are deposited on the surface of the meat and are therefore killed when the meat is cooked, I should like to point out that it is quite a usual custom for shop butchers to remove the bones from a joint of meat which has been sold and roll the meat and secure it with wooden skewers. When this is done the contaminated surface in most cases is inside the roll and may not be brought to a sterilizing temperature during the subsequent cooking process, if the purchaser happens to be partial to "underdone" meat. Even if the contaminated surface were on the outside of the roll, many of the tubercle bacilli would probably be carried deeply into the meat by the insertion of the wooden skewers. Furthermore, it has to be remembered that some of the meat from a contaminated carcass will probably reach the consumer in the form of sausages and, as is well known, these are often consumed in a semi-raw condition.

All this shows the need for efficient supervision and inspection wherever animals are being slaughtered and dressed for human food. If the high standard of inspection existing at the State Abattoir could be applied to the whole of New South Wales, the people of this State would have no need to be concerned regarding the wholesomeness of their meat supply.

The standard of meat inspection in Germany and certain other countries is modified by the fact that meat at a price which the poorer people can afford to pay, is never very plentiful and this has led to the establishment of the *Freibank* system which is now an indispensable factor in the meat inspection and meat supply services of these countries.

In Germany much of the meat that would be condemned under our system or under the American system, is sterilized by steam under Government supervision after the actual lesions have been removed and it is then sold to the public at a low price through the local *Freibank* which are special shops or places set apart for the sale of this class of meat. This system works well in Germany where it was legalized as long ago as 1879.

The adoption of the *Freibank* system in Australia is neither necessary nor practicable at the present time, as the public would refuse to buy meat which required sterilization before sale.

In regard to the incidence of bovine tuberculosis in man, it seems to me that opinions have been based

on insufficient data. According to Moller the literature up to 1916 cited only 2,051 cases of tuberculosis in man in which the tubercle bacilli were typed. In 189 instances bacilli of the bovine type were found; in 1,848 bacilli of the human type were responsible, while in fourteen both human and bovine types were involved. Of the 189 cases of purely bovine type of infection 151 occurred in children and in 114 the disease was considered to have resulted from alimentary infection.

These figures show that the bovine type of tubercle bacilli was found in nearly 10% of the cases and of these one-fifth occurred in persons other than children.

When one considers the enormous numbers of fatal and non-fatal cases of tuberculosis in man that have occurred throughout the world, including the large number of people now living that are affected with the disease, it does seem reasonable to suggest that the relatively small amount of investigational work which has been done on the incidence of bovine tuberculosis in man, is far too small to justify any claim to a real knowledge of the facts.

Among the most interesting cases cited in the literature, to my mind, were the ten of mixed or double infections quoted about the year 1912 by Park and Krumwiede, of New York, in a summary of infections investigated by other workers. These were of unusual interest, not only in regard to the various ages of the patients concerned, but also because of the location of some of the lesions produced by the bovine type of bacilli.

The essential details are as set out in Table II.

TABLE II.

Lesion.	Time.	Type of Infection.
Pulmonary tuberculosis	20 years	Sputum, human and bovine types.
	27 years	Sputum, human and bovine types.
Abdominal tuberculosis	70 years	Mesenteric glands, human type. Retroperitoneal glands, human and bovine types.
Generalized tuberculosis (alimentary origin)	5½ years	Spleen, human type. Mesenteric gland, bovine type.
	18 years	Lung (culture not obtained). Mesenteric glands, human and bovine types.
	30 years	Bronchial glands, human type. Mesenteric glands, human and bovine types.
Generalized tuberculosis	9 months	Bronchial glands, bovine type. Mesenteric glands, human type.
Generalized tuberculosis, including meninges (alimentary origin)	4 years	Meninges, human type. Bronchial glands, human type. Mesenteric glands, bovine type.
Generalized tuberculosis, including meninges	4 years	Bronchial glands, spleen (injected together), bovine and human types.
	4 years and 7 months	Meninges, human type. Lung, human type. Mesenteric nodes, human type. Bronchial nodes, human and bovine types.

While much may be said in favour of the Pasteurization of milk, it must not be forgotten that it cannot always be relied upon to give protection against infection with tubercle bacilli even under laboratory conditions. This was demonstrated by Anderson (*Maanedsskrift for Dyrlaeger*, Volume XXXVI, 1924).

Ten experiments were carried out and in three of them the tubercle bacilli survived the Pasteurization process (heating to 63° C. for half an hour) and samples inoculated into guinea-pigs produced fatal tuberculosis.

TUBERCULOSIS OF ANIMALS IN RELATION TO PUBLIC HEALTH.[1]

By H. R. CARNE, B.V.Sc. (Sydney),
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IT has been recognized that of the tuberculous infections in man contracted from animals, those due to the bovine type of the tubercle bacillus are the most common. Such infections usually result from the ingestion of infected milk. It must be remembered, however, that some of the domestic animals and some birds are susceptible to the human type of bacillus and may be responsible for infection of humans.

With regard to the avian type of tubercle bacillus, statistics up to the present have brought to light only a very small number of authenticated cases. Van Es states that in European literature appearing between 1893 and 1923 there are accounts of fourteen cases of avian infection of man that have been definitely ascertained by typing experiments. Recent work on Hodgkin's disease, however, has raised considerable interest in the possibility of the avian tubercle bacillus being the cause of this condition. Élise S. l'Esperance has recently reported that chickens, inoculated intravenously with emulsions of lymphatic glands from two patients with Hodgkin's disease in man, developed either typical or atypical tuberculosis. In tissue smears of the experimental lesions, acid-fast granules and rods were demonstrated. Reinoculation of material from these experimental lesions into another bird produced the same but more severe lesions. Atypical tuberculosis was produced in guinea-pigs by inoculation of tissue from lesions in the fowl. A pure growth of bacteria showing the characteristics of the avian tubercle bacillus was obtained on egg media sown with material from lesions in the guinea-pig. Again, Hudelo, Rabut, Kaplan and Ragonneau have reported two cases of Hodgkin's disease in man which terminated in acute tuberculosis.

It is of interest, therefore, to review briefly the occurrence of tuberculosis in domestic animals, noting particularly the types of tubercle bacilli which are responsible for the disease in the various species.

Food Animals.

Pig.

The pig is very commonly affected. It is susceptible to bovine and avian strains and slightly

[1] Read at a combined meeting of the New South Wales Branch of the British Medical Association and of the Veterinary Association of New South Wales on November 28, 1929.

susceptible to the human strain. It is most commonly affected with the bovine strain and the occurrence of tuberculosis in pigs is usually a good indication of the presence of tuberculosis in the dairy herd from which their food supply is drawn.

It has been recently found that quite a considerable percentage of tuberculous infections in pigs is due to the avian type of bacillus. Stanley Griffith has recorded that of 163 tuberculous pigs examined by English investigators, 43 were infected with the avian type. Such infections have in all probability have been contracted by the ingestion of material contaminated by fæcal matter from fowls which are commonly allowed to run in pig sties. Helm found that 80% of pigs were susceptible to avian tubercle bacilli and Raebiger and others have given a detailed description of the disease produced experimentally in swine. Efficient meat inspection and the usual processes of cooking render it unlikely that pigs infected with either bovine or avian strains are a danger to human health.

Sheep.

Sheep are very rarely affected. They are susceptible to bovine and avian strains.

Cattle.

The large majority of infections in cattle are of the bovine type, but cattle are susceptible to the avian type also. Van Es states that among 113 tuberculous infections in cattle investigated at the Nebraska Experimental Station in the United States of America, ten were due to the avian type and four were mixed avian and bovine. The occurrence of avian tuberculosis in cattle has also been reported from England and Denmark. Milk containing the bovine organism is the common source of infection of man, the danger from meat being negligible where efficient meat inspection exists.

Poultry.

Tuberculosis of poultry is very common in certain districts of some countries. For example, Raebiger reported that in Saxony 54% of flocks in towns and 85% in villages are infected. Lesions are chiefly abdominal and the causal bacilli are frequently excreted in the fæces. It has been shown by a number of different investigators that living tubercle bacilli may be present in fowls' eggs, but Fitch and his coworkers concluded that less than 1% of the eggs from tuberculous fowls contains tubercle bacilli and thus it is probable that the egg is not of any great importance in the transmission of avian tuberculosis.

Working Animals and Pets in Contact with Man.

Horse.

The horse is not commonly affected. When it is infected the infection is caused by the bovine strain. The horse is an unlikely source of infection of man.

Dogs.

Dogs are susceptible to both human and bovine strains. They are commonly affected with the human type. Open lung lesions are a source of danger for humans.

Cats.

Cats are very susceptible to the bovine strain, but highly resistant to the human.

Parrots.

Tuberculosis is seen only in those species of birds which live in close contact with man or cattle. According to Calmette it is never seen in wild birds in their free state.

Parrots are usually affected with the avian type, but may be affected with the human type. Cutaneous lesions which are rare in lower animals, may occur in the form of greyish, dry, scaly growths on the head or feet and such birds may be a source of infection for man.

Infected Milk.

The percentage of cattle infected with tuberculosis which have actual lesions of the udder, is relatively small. The most recent statistics on this point are those of Chesley, of Minnesota, United States of America, who reports that of 1,200 autopsies on cows which reacted to the tuberculin test, tuberculosis of the udder was found in 5·75%. Where tuberculosis was generalized, the udder was affected in about 17% of animals.

Milk from a tuberculous udder may contain enormous numbers of tubercle bacilli, instances of 100,000 per cubic centimetre having been recorded. Delépine showed that one part of milk from a cow with tuberculosis of the udder when diluted in 100,000 parts of non-tuberculous milk, is capable of infecting guinea-pigs and therefore one cow with a tuberculous udder is capable of infecting the mixed milk from a large herd.

The dose of tubercle bacilli ingested is important and thus the great danger of infected individual family cows where the factor of dilution is absent.

A most important observation which has been confirmed by English, French, American, German and Japanese workers, is the fact that cattle infected with tuberculosis but whose udders were free from any demonstrable lesions, are able to excrete bacilli in the milk. Thus any tuberculous cow is a potential source of infected milk.

Filterable Forms of the Tubercle Bacillus.

In recent years filtration of material from lesions of tuberculosis through porcelain candles which retain visible bacteria, and inoculation of the filtrate into guinea-pigs have been reported by various workers to give rise to the development of lesions in which acid-fast bacilli can be demonstrated. Although such lesions are not usually those typical of infection with fully virulent bacilli, considerable evidence has been brought to support the opinion that the tubercle bacillus is able to assume ultramicroscopic and filterable forms. The possibility of such filterable forms being present in milk should be kept in mind.

Pasteurization.

Pasteurization has proved its value in reducing the occurrence of milk borne epidemics due to typhoid, paratyphoid and diphtheria bacilli and in

all large towns and cities in which the milk supply has to be drawn from considerable distances, particularly in hot weather, Pasteurization is of great value in increasing the keeping qualities of milk where the hygiene of milk production and handling is not of a high degree of efficiency. Pasteurization, however, has a number of objections amongst which is the fact that "dirty" milk with a high bacterial content may not be rendered innocuous, as the sporing putrefactive bacteria are not destroyed nor are the heat resistant toxins of the paratyphoid organisms.

With regard to the destruction of tubercle bacilli in milk during Pasteurization, many of the experiments to determine the requisite temperature for the destruction of the tubercle bacillus have been carried out with milk infected with tubercle bacilli grown on artificial culture media.

J. L. Meanwell, of the National Institute of Research in Dairying, has recently investigated the effect of Pasteurization on tubercle bacilli in naturally infected milk. He found that an exposure of thirty minutes to a temperature of 62·8° C. (145° F.) which is the temperature usually employed in Pasteurization by the holder process, does not invariably kill the tubercle bacilli. At a temperature of 60° C. (140° F.) for twenty minutes tubercle bacilli are destroyed in many cases, but this combination of time and temperature leaves no margin of safety.

These observations together with the technical difficulties associated with the maintenance of an even temperature, avoiding foam formation and surface cooling *et cetera,* emphasize the importance of keeping a very close check on the efficiency of Pasteurization plants.

Vaccination Against Tuberculosis in Animals.

The numerous methods of preventive vaccination against tuberculosis and their disappointing results are well known. Recently, however, French workers have claimed most encouraging results with the so-called *Bacille Calmette Guérin* vaccine which consists of a living culture of bovine tubercle bacilli which has been attenuated by subcultivation over a period of thirteen years on potato medium containing glycerinated ox bile. This strain has lost the power of giving rise to progressive lesions of tuberculosis in guinea-pigs and other susceptible animals. The inoculated bacilli appear to remain *in situ* or in the neighbouring lymphatic glands. This vaccine has been extensively used on children and investigations have also been carried out on cattle. Guérin, Richard and Boissière reported in 1927 the results of vaccinating all newly born calves in a herd which at the beginning of the experiment contained 41·7% of animals which reacted to tuberculin. The hygiene and general management of the herd were not altered in any way and it was claimed that all vaccinated animals, slaughtered in their prime for beef, were free from lesions of tuberculosis.

Harnach also reported favourable results with experiments on fowls which were vaccinated with a similarly attenuated avian strain.

These investigations raised considerable interest in English speaking countries where the value of this method of vaccination has been tested. The results have been uniformly unfavourable. Thus Woodruff and Gregory in Australia, Larson and Evans, Schroeder and Crawford in the United States of America have found that in cattle which had been vaccinated with *Bacille Calmette Guérin* according to the French method, any immunity which might have been produced, was evanescent and the animals were found subsequently to be susceptible to virulent bacilli which were introduced into the body by various routes.

Not only has this method of vaccination failed to give the protection which has been claimed, but, further, it presents the very serious disadvantage that vaccinated animals become sensitized to tuberculin and give a positive reaction with the tuberculin test. This would interfere with any method of control or eradication where the tuberculin test was employed as a means of detecting infected animals.

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BOVINE TUBERCULOSIS IN MAN.[1]

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DR. BARET'S paper has been perused with interest and although one would hesitate to do so, it would almost seem that one would be justified in describing it as of the nature of a special pleading.

In dealing with it from the point of view of field control, it appears evident that the necessity for such field control should be first considered. The methods by which such field control may be carried out, are fairly obvious and will be referred to later.

In the first place it is desired to stress the fact that in all work carried out in connexion with tuberculosis in cattle by the Department of Agriculture of New South Wales, two points have been emphasized, first, that the situation in Australia is far better than in England or Europe and, secondly, that anything in the nature of panic action is to be deprecated, as it would be more likely to defeat its own end than to bring about any good. This, however, is far from saying that continued and increased effort against the disease is not required.

Dr. Baret has produced a series of figures based on abattoir inspection, from which he concludes that the incidence of tuberculosis in New South Wales cattle has declined very considerably in the last ten years. It may be accepted that a decline has taken place, but Dr. Baret has neglected to take into consideration certain important factors, first, the age at which cattle are now slaughtered, for that is on the average lower than it was even ten years ago, whilst the percentage of Queensland cattle slaughtered has been higher. Both factors are liable to operate in the direction of lowering the incidence of tuberculosis in the cattle slaughtered and particularly in lowering the percentage of those condemned, for it must be borne in mind that, in comparing the statistics given from Sydney and Newcastle abattoirs with the figures he gives comparing the City of London and the City of Edinburgh, there is a very considerable difference. His figures for Sydney and Newcastle deal only with condemnations; the figures given for London and Edinburgh show the percentage tuberculous and these are obviously two very different things. If the percentages in London and Edinburgh are looked at, it will be noted how heavy is the percentage of cows found tuberculous, that is, the older the animal the higher the percentage of tuberculosis. This is a point of very great importance in connexion with tuberculosis on dairy farms.

Dealing with the results of the tuberculin test, Dr. Baret quotes certain instances, but first of all refers to 1,730 dairy heifers purchased for the Western Australian Government, of which 3·1% reacted. It is to be noted that these were young animals and if the figures for Edinburgh and London are observed, it will be noted what a difference there is in the percentage affected amongst heifers and the percentage affected amongst cows. Moreover, these heifers were inspected before testing, consequently it is safe to assume that no clinical cases were included amongst them, that is, they were to an extent selected animals.

Dr. Baret then states that Mr. Symons, Chief Veterinary Surgeon, tested 1,163 cattle and 3·4% reacted. It is not known whence these statistics were obtained, so it is not possible to criticize, but it is followed by the statement that the present Chief Veterinary Surgeon tested 1,061 dairy cows and 5·4% reacted. It is presumed that this refers to cows tested by the veterinary staff of the Stock Branch in connexion with the accredited tuberculosis-free herd scheme. If the two figures are really comparable, there would appear to have been an increase in tuberculosis amongst the dairy cows. It is doubtful, however, if they are comparable. So far as the herds tested under the tuberculosis-free herd scheme in New South Wales are concerned, it is obvious that they cannot be taken as a general reflex of the condition of the cattle throughout the State. Some of the larger herds on that list were herds belonging to Government institutions in which a very considerable amount of testing, in some cases up to the point of cleansing, had been previously carried out within recent years before the introduction of the scheme. Others amongst the big herds belong to exceptionally careful men who had taken considerable pride in possessing cattle of good type and in good health. Moreover, very few of the herds are in the real dairying districts. Other herds were from 30% to 50%. Naturally these are not yet tuberculosis-free.

The statement is made that in England it is generally said that 33% of the dairy herds are tuberculous in some form or other. It would be a bold man who would say regarding any dairying country, except those in which very extensive testing had been carried out and elimination of reactors had taken place, that 66% of herds were not tuberculous in some form or other.

With regard to the investigations into the presence of tubercle bacilli in the metropolitan milk supply, there is one point which is not clear. Were

[1] Read at a combined meeting of the New South Wales Branch of the British Medical Association and of the Veterinary Association of New South Wales on November 28, 1929.

the 348 samples examined by the Dairy Farmers' Co-Operative Milk Company, Limited, simply microscopically examined or were they confirmed by biological test? It would be extremely difficult to detect tubercle bacilli in a sample drawn from a bulk supply representing anything from sixty to two hundred suppliers. If it were possible to detect tubercle bacilli on smear examination in such a way, the condition of the milk supply would be something appalling.

Dr. Baret suggests that as the majority of cows that react, manifests only small lesions of tuberculosis which could not possibly have any influence in affecting the milk, the necessity for action in the case of these reactors is not obvious. It is, of course, a fact that many cows that react, manifest very small lesions, but the point of importance is that it is impossible to say when the lesion of any infected cow will make it capable of transmission.

The question is asked whether in cattle a great majority of reactors conquers the disease. By conquering the disease is presumably meant either the encapsulation of lesions or the calcification of lesions. Judging from *post mortem* results, the occurrence of either of these changes is very infrequent in cattle.

The tables dealing with the incidence of tuberculosis in man are very satisfactory up to a certain point. They do reveal a marked diminution in the death rate from this disease and it is reasonable to assume that some of this is due to the increased attention given to the health of dairy cattle. Dr. Baret's table VII, showing the number of deaths from tuberculosis in children under five years of age, is of particular interest. It shows to what a comparatively slight extent pulmonary tuberculosis is responsible for this mortality and how all through non-pulmonary tuberculosis has been a serious factor. It is just this non-pulmonary tuberculosis which is so often of bovine origin. The fact that it has diminished, as it has, is an indication of satisfactory work. The fact that it has not been eradicated is an indication that the work has not gone far enough and if it is possible to expedite the elimination of non-pulmonary tuberculosis by any means, much may be said in favour of action which would bring about that result. It has also to be borne in mind that over and above the cases in which death is caused from non-pulmonary tuberculosis, there are those numerous cases in which diminution of efficiency can be ascribed in part at least to the incidence of the disease.

It is satisfactory to note that Dr. Baret does not consider that the improvement which has taken place, is any reason or excuse for not attempting to go further.

Dr. Baret then states that the first precaution is Pasteurization. Now the subject of Pasteurization is a very vexed one. Its benefits are freely recognized and have been widely spoken of; its defects have not been given equal consideration. Too much confidence is placed on the efficacy of Pasteurization. As instances of what may be considered the defects of Pasteurization, the following points may be considered:

Dr. Baret adds: "I do not know of any experiments that proved raw milk to be of greater value than Pasteurized milk." Recent work has indicated that in some respects Pasteurized milk is not equal to raw milk.

Dr. Magee, of the Rowett Institute of Aberdeen, is quoted by the *Veterinary Record* of July 13, 1929, as having read a paper for the Zurich Congress of the Royal Institute of Public Health on some effects of heat on the nutritive properties of milk. Unfortunately the paper is not to hand, but the *Veterinary Record* states that the paper was illustrated by lantern slides and emphasized the deleterious effects of heat on the calcium and iodine content of milk.

An interesting series of experiments was carried out by Kramer, Latzke and Shaw on a comparison of raw, Pasteurized, evaporated and dried milks as sources of calcium and phosphorus to the human subject, from which the following is quoted:

> From experience with five children it is evident that the child retains more calcium when it is supplied in fresh milk than when it is furnished in equal amounts by dried milk, other factors remaining unchanged. All adult subjects showed more favourable calcium balances when fresh milk was the source of supply rather than dried milk. Pasteurized milk also gave less favourable calcium balances than did fresh milk.

Magee and Glennie, reporting in the *Biochemical Journal* for 1928 stated that heat caused the disappearance by volatilization of 20% or more, according to the duration of heating, of the total iodine of separated milk. The ratio of diffusible to non-diffusible iodine was the same in the milk after heating as it was before.

In the *Scottish Journal of Agriculture*, October, 1926, Orr, Creighton, Haldane and Middleton gave the results of a series of experiments with pigs fed on rations to which was added either fresh milk, Pasteurized milk or sour milk. The graph which accompanies this article is extremely interesting. For the first six days the animals were given no milk. On the introduction of milk to the ration the rate of absorption of calcium, phosphorus and nitrogen rose rapidly and as the rate of retention of these elements is the indication of the rate of growth of bone and muscle, that part of the graph forms an excellent illustration of the value of milk for growing animals. As soon as the Pasteurized milk was substituted for the fresh milk, the rate of retention dropped rapidly, particularly as regards calcium and phosphorus, and then began to rise again slightly. When sour milk was substituted for the Pasteurized milk, there was an immediate rapid rise in the retention of these elements.

Some very interesting work will also be found in the *Scottish Journal of Agriculture* on the results of feeding tests with milk and the conclusions drawn on these were that heat reduced the amount of soluble calcium in milk; the absorption and retention by growing animals of calcium, phosphorus and nitrogen is lower on a diet of Pasteurized milk than on a similar diet with the same amount of fresh

milk; the addition of a soluble calcium salt to Pasteurized milk fed to growing animals increases the amount of calcium, phosphorus and nitrogen absorbed and retained; in feeding experiments with calves the rate of growth was slightly less on Pasteurized milk than on fresh milk or milk not Pasteurized *plus* calcium lactate.

There is a very considerable amount of evidence to the effect that vitamin *C* is destroyed or the vitamin content lowered by Pasteurization and still more by boiling.

It seems, therefore, that there is a considerable amount of evidence to the effect that Pasteurized milk is not so valuable as fresh milk and in view of the recent high importance which has been ascribed to iodine, phosphorus and calcium and which is certainly true regarding phosphorus and calcium, then the effect of heating on milk must be given serious consideration.

The action of heating on the proteins contained in milk is not dealt with because it is so obvious and yet is probably not affected by ordinary Pasteurization, though it does appear to be so affected by boiling.

There is also a point in connexion with Pasteurization which is likewise obvious, and that is the question of the keeping capacity of milk after Pasteurization. With milk which has not been Pasteurized, souring can be taken as a general indication of the conditions under which the milk has been kept, its age and its present state, but under Pasteurization souring is so definitely interfered with that it is difficult to judge the condition of the milk and there may be considerable bacterial contamination which results in undesirable changes in the milk, but which does not cause changes obvious to the persons using the milk.

The efficacy of Pasteurization has also to be considered. Dr. Baret states that the concensus of opinion seems to be that milk should be Pasteurized at a temperature of 60° C. (140° F.) by a heating process for thirty minutes. He points out, however, that as Pasteurizing plants may fail either from a mechanical or through the human element, it will still be essential by thorough dairy inspection to continue to educate the dairyman in sanitary methods. In view of the fact that Pasteurization may fail, as indicated, would it not be also desirable to continue and to extend, if possible, the efforts made to free the herds from tuberculosis.

A considerable amount of work has been done on the effect of Pasteurization on the bovine tubercle bacillus of milk. Meanwell's work is quoted in the *Veterinary Record* for April 7, 1928. His conclusions are: (i) A temperature of 62·8° C. (145° F.) for thirty minutes does not invariably kill the tubercle bacillus in naturally infected milk, although in most cases this temperature is effective. (ii) At a temperature of 60° C. (140° F.) for twenty minutes tubercle bacilli are in many cases destroyed, but this combination of time and temperature leaves no margin of safety.

Whilst on the subject of Pasteurization I may quote W. G. Savage in *The British Medical Journal*, September 14, 1929. In dealing with measures directly aimed at reducing the amount of tubercle bacilli in milk, he uses the following terms:

> There are very considerable objections to a compulsory system of Pasteurization on other grounds related to the various problems of clean milk, and if we have to adopt it as the best available method of protection from tuberculous milk, I should regard it as a confession of failure and the acceptance of the second best procedure, only to be adopted with very adequate proportions. . . . In view of these drawbacks and other inherent objections to Pasteurization, I greatly prefer the adoption of the protective measures indicated to deal with bovine tuberculosis, but if these are not attainable, Pasteurization seems less of an evil than the present tinkering procedure.

It would therefore appear:

1. That tuberculous milk is a danger to child life.
2. That that danger is far less in Australia than in Great Britain and Europe.
3. That reliance on Pasteurization as the main standby in protecting child life against bovine tuberculosis is not desirable.

It should, I think, be stated definitely that the ideal milk supply is one of fresh milk from tuberculosis-free cattle. Whilst it may be necessary to adopt Pasteurization for the milk supply of large cities at present as a second best alternative, continually increasing efforts should be made to bring about such a state of affairs as will permit of a copious supply of fresh tuberculosis-free milk.

The question of field control of disease naturally enters very largely into the development of this ideal. Field control may embrace a number of different measures: (i) The routine examination and the detection of clinical cases by visual and manual examination; (ii) the testing by microscopical and biological methods of the milk supply from separate herds in order to determine whether at that particular time the milk is free from tubercle bacilli; (iii) the creation of tuberculosis-free herds from which the milk is sold for specific purposes; (iv) the gradual extension of this system until it embraces areas; (v) continued attention to the sanitary conditions under which cattle are kept; (vi) the detection of tuberculous herds through the operations of such legislation as the *Swine Compensation Act* of New South Wales.

The aim of all these measures is to reduce the incidence of tuberculosis in cattle and consequently to reduce the possibility of infection from the milk. The visual examination of cattle is valuable to a point, but unfortunately it is apt to miss the most dangerous cow of all, that is the cow with a tuberculous lesion of the udder which is not definitely visible, whilst at the same time it brings about the destruction of a number of cattle which are at that particular time of no greater danger to the milk supply than are many of the reacting cattle showing no clinical symptoms. This is a point which is often overlooked and which the opponents of the application of the tuberculin test consistently ignore. This is not to say that visual examination is not valu-

able. It obviously results in removing from the herds a number of affected cows any of which may at any time become capable of transmitting the disease. The manual examination of udders takes the control considerably further, as it does assist in detecting the most dangerous cow, and if such manual examination is followed up by the application of the tuberculin test in suspicious cases, very useful work may be accomplished. It has to be noted, however, that although this system has been in existence in Victoria for a considerable period, yet according to recent work milk containing tubercle bacilli was to some slight extent reaching the Melbourne market.

It may be held that neither visual nor manual examination will eliminate tuberculosis, but it is an important factor in reducing its incidence. The routine examination of milk from individual farms frequently enables the veterinary officer to proceed straight to the source of tuberculous milk and there by examination and elimination to determine to some extent which animals are involved. Clinical examination and the application of the tuberculin test will then come into play to eliminate the dangerous animals. In the same way tracing up tuberculous pigs to their source of origin will frequently enable the cows excreting tubercle bacilli to be detected and removed.

Whilst all this work is valuable, it still leaves in the herds animals which at any time may commence passing tubercle bacilli in the milk, and the only satisfactory way of preventing that possibility is the application of the tuberculin test to the whole herd.

In America and Canada the tuberculin test has been used to an enormous extent in cleaning up the herds; the United States has tested something like 30,000,000 cattle and about 1,000,000 reacted. The Canadian system aimed at the cleaning up of certain areas and appears to have been particularly successful. A small start has been made in New South Wales by the establishment of tuberculosis-free herds.

The work in most of the Australian States requires coordinating and concentrating in order to do away with divided effort.

The economic side of the question has not been touched, although it is in many cases of very considerable importance to the stock owner, but hardly touches the question under consideration.

OBSERVATIONS ON THE MORTALITY FROM HEART DISEASE IN THE STATE OF NEW SOUTH WALES.

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THE rapid and progressive increase in the number of deaths attributed to heart disease in the mortality statistics of New South Wales, both absolutely and relatively to the population, is sufficiently striking to arrest attention. To illustrate the fact Table I has been compiled from the reports of the Government Statistician of this State.[1] For the sake of comparison the number of deaths and the rates per 10,000 living of the population from the eight principal causes of death have been tabulated. The table shows the figures for individual years from 1902 to 1922 at intervals of five years and also the figures for the years 1922 to 1928 inclusive. The numbers represent the number of deaths in each year; the rates represent the proportion of deaths per 10,000 living. For the purposes of this review the following features are to be noted:

1. The rapid and progressive increase in the number of deaths due to heart disease. In 1928 they exceeded the combined deaths due to cancer and tuberculosis or any other two combinations with the exception of cancer and pneumonia which were slightly in excess.

2. The number of deaths has quadrupled while the rate has doubled.

3. The number of deaths due to old age is decreasing rapidly.

TABLE I.

Deaths and Death Rates of Eight Principal Causes of Death. State of New South Wales.[1]

Year.	Heart Disease.		Cancer.		Pulmonary Tuberculosis.		Pneumonia.		Nephritis.		Apoplexy.		Accident.		Old Age.	
	Number.	Rate.	Number.	Rate.	Number.	Rate.	Number.	Rate.	Number.	Rate.	Number.	Rate.	Number.	Rate.	Number.	Rate.
1902	972	7·00	869	6·26	1,152	8·30	1,351	9·73	561	4·04	385	2·77	990	7·13	995	7·17
1907	1,427	9·40	1,085	7·15	976	6·43	1,048	6·90	639	4·21	585	3·85	841	5·54	949	6·25
1912	1,881	10·79	1,312	7·52	1,078	6·18	1,098	6·30	923	5·29	611	3·50	1,027	5·89	1,263	7·24
1917	2,204	11·57	1,491	7·83	1,017	5·31	1,063	5·58	987	5·18	594	3·12	973	5·11	1,109	5·82
1922	2,501	11·63	1,871	8·70	1,080	5·02	1,355	6·30	1,007	4·68	658	3·06	939	4·37	1,009	4·69
1923	2,729	12·45	1,879	8·57	1,114	5·08	1,701	7·76	985	4·49	669	3·05	949	4·33	1,214	5·54
1924	2,756	12·36	2,078	9·32	1,165	5·22	1,511	6·78	1,103	4·95	671	3·01	1,072	4·81	1,054	4·73
1925	3,067	13·48	2,133	9·37	1,923	4·49	1,545	6·79	1,119	4·92	873	3·84	1,121	4·93	1,078	4·74
1926	3,280	14·13	2,157	9·29	1,144	4·93	1,608	6·93	1,108	4·77	1,027	4·42	1,255	5·41	1,073	4·62
1927	3,785	15·94	2,210	9·31	1,056	4·45	1,929	8·12	1,140	4·80	1,060	4·46	1,415	5·96	1,048	4·41
1928	3,849	15·86	2,280	9·40	1,166	4·81	1,715	7·07	1,245	5·13	804	3·31	1,284	5·29	784	3·23

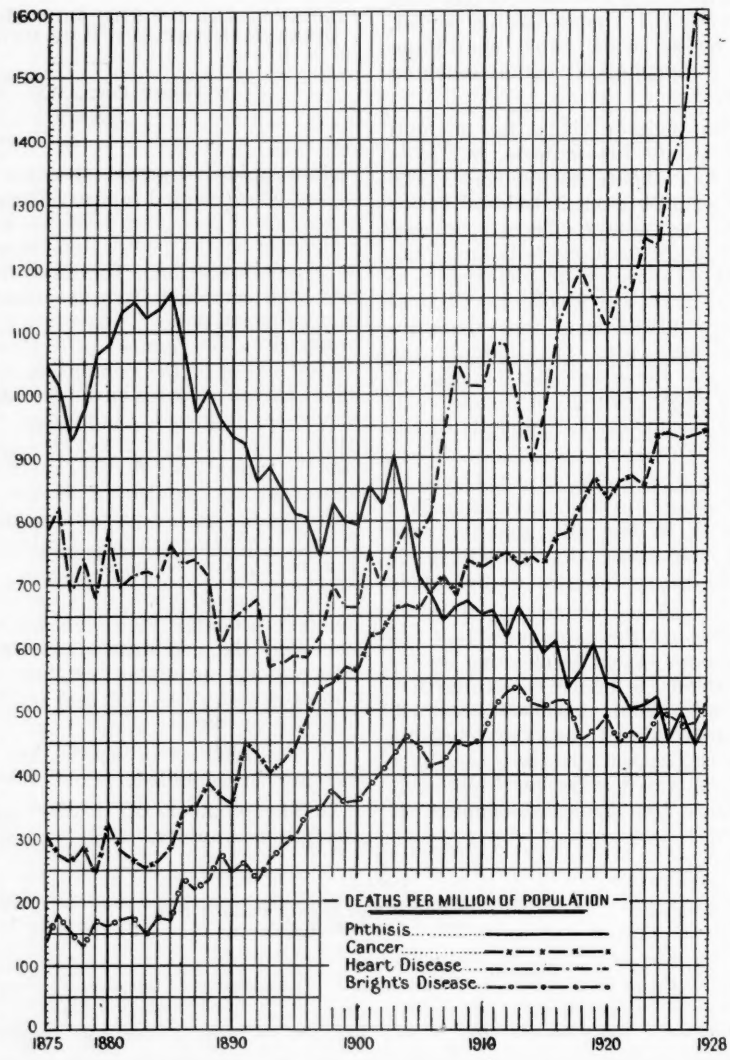
[1] The deaths are in numbers of the total population. The rates are the rates per 10,000 living.
In 1902 the mean population of the State was 1,388,400.
In 1928 " " " " 2,426,300.

4. The mean population has increased from 1,388,400 in 1902 to 2,426,300 in 1928.

Figure I is a chart taken from the Official Year Book of New South Wales, 1928. It is self-explanatory.

The Object of the Investigation.

The primary object of this study was to determine, if possible, the cause of the increase. It was not expected that any ætiological factor would be determined for the reason that mortality statistics do not yield such information. An attempt has been made, in view of the high mortality rate of heart disease, to determine whether heart disease is actually on the increase or whether the figures, as such, truly represent the position so far as heart disease is concerned.



The Value of Statistics.

Statistics is defined as "the science of the collection of facts on the basis of relative numbers or occurrences, as a ground for induction of facts or for inference of general truths"; also as "classified facts respecting the condition of various respects of the people in a State or respecting any particular class or interest."

With regard to the particular case of heart disease it is hoped to show that for several reasons which will be discussed, the inferences to be drawn from the figures tabulated are not so accurate, as for example those for cancer or tuberculosis.

Heart disease presents special difficulties from a public health point of view. It has no pressing needs as a rule. Compared with cancer, for example, it has few of the distressing accompaniments. It does not present the urgent call for immediate attention that exists in regard to diphtheria, scarlet fever *et cetera*. Ten or fifteen years may elapse before the disease forces itself on the attention. It is frequently manifested as a terminal event only. Heart disease varies widely in its nature and is produced by such a diversity of causes that the problem of evaluating the influence of the several causes is complex and difficult. It will be shown that rightly or wrongly it appears as a cause of death and is given preference in mortality tables in a number of cases where its presence is often assumed or given as a joint cause of death it is tabulated as a death due to heart disease.

The Information to be Gained from Mortality Tables.

While the failure of mortality tables to indicate ætiology is true, it is nevertheless a fact that, by showing the number of deaths due to a particular disease, attention is attracted to that disease; inquiry is stimulated to account for the increase or decrease, as the case may be. It is due to the fact that the mortality tables showed increased death rates in the case of tuberculosis and infantile diseases, to mention only two, that the vigorous campaign of modern times against them have been instituted. The same applies to cancer today. Here the increase in the number of deaths, coupled no doubt with the dread nature of the disease in the public mind, a point of distinction in this respect from heart disease, is responsible for the world-wide endeavour to stem its tide. In passing it is interesting to note that the recent investigations of Dr. Cherry with regard to cancer began as the result

of statistical observations on the relationship between the sum of deaths from cancer and tuberculosis, the rise in death rate from the former being accompanied by a fall in the latter. Further, the results of the campaigns and the remedies used in combating disease may be measured with some accuracy by mortality tables.

Difficulties in Certification and Tabulation.

There are many difficulties in the correct certification and tabulation of causes of death and in the case of heart disease in particular these difficulties are perhaps greater than in any other. They have been so aptly expressed by the late Sir George Knibbs[2] that his remarks are quoted in full.

> With a complex organism such as the human body the ætiology of disease and death is ordinarily by no means a simple matter, though in popular statements it may appear to be so. Many minds are satisfied when a mere name is given as a cause. Excepting death from external violence, probably life's termination is always due to a complex of conditions and the assigning of one of these is in a measure arbitrary and empirical. For example the toxins developed by certain conditions may so impair the heart's action that more than ordinary care as to physical effort is indicated as necessary, but undue strain being put on it, the result is death. The immediate cause of death is the undue strain on a weak heart; the complex cause is the toxin impairment together with the undue strain. Or again, suppose that as a consequence of syphilis, arteriosclerosis of the brain exists and by some action the blood pressure is greatly increased, resulting in death from apoplexy. The primary cause is the syphilis; the final cause is the consequence of the extravasation of blood; the arterio-sclerosis is but a link, as it were, in the chain of causes or rather coexistent conditions.
>
> These two examples will perhaps sufficiently illustrate the point that in order to be practically serviceable a classification of diseases and causes of death must necessarily abandon any attempt at rigorous exactitude and rest satisfied with rules of procedure which roughly serve the main purpose of the classification. It is self-evident that a classification for one purpose will probably have limitations from the point of view of another purpose. It may be noted, therefore, that the existing international classification has been developed by men who were both medical practitioners of high reputation and statisticians of eminence. Moreover, at each revision the experience gained by its use during the intervening period has influenced the changes made. A study of these will convince any methodologist that the improvements made have been very great.

And again:

> In all parts of the world those who have given special attention to the statistical records of causes of death, seem satisfied that they can be of great service in studying the incidence of disease on humanity and the amelioration of the various conditions. . . . If the records are kept permanently, it is obviously possible to renew them in the light of subsequent discoveries in the nature of disease.

A perusal of the whole address will show the difficulties of certification and tabulation and the remedies proposed to combat them.

Meaning of the Term Heart Disease.

Before drawing any deductions from the figures as tabulated in Table I, it is necessary to explain what is meant by the term heart disease. Under the classification of diseases, Bertillon system as revised by the International Commission, Paris, October, 1920, which is in world-wide use and is the system adopted in this State, heart disease comes under the heading of Classification IV, diseases of the circulatory system. The term heart disease includes deaths from pericarditis (acute or chronic, rheumatic *et cetera*), endocarditis and myocarditis (acute), *angina pectoris* and other diseases of the heart. The latter include aortic valve disease, mitral valve, aortic and mitral and other valve diseases, fatty heart, cardiac dilatation, other or unspecified myocardial disease, disordered action of the heart, heart disease undefined. The figures in the above table, therefore, cover the deaths resulting from any of the above mentioned diseases. They do not include deaths from any disease of the circulatory system such as aneurysm, arteriosclerosis, embolism, thrombosis *et cetera*. Deaths from these causes represent roughly as a rule 12% to 18% of those due to heart disease. Thus in 1926 the total number of deaths due to disease of the circulatory system was 3,739. Of these 3,280 were due to heart disease and 459 to other diseases of arteries *et cetera*. In 1928 there were 4,705 deaths under diseases of circulatory system; 3,849 were due to heart disease and 856 to disease of arteries *et cetera*. Mention may be made here of the fact that in 1928 of the 856 deaths not due to heart disease 683 were due to arteriosclerosis. Of these 320 were due to arteriosclerosis with record of a cerebral vascular lesion and 363 (total 683) to "arteriosclerosis without record of a vascular lesion." Deaths from aneurysm, 66, and other diseases of arteries, 32, amounted to 98. The figures are of interest as showing the high incidence of vascular disease and are worthy of consideration in connexion with the mortality rates of heart disease, for the reason that arteriosclerosis stands as a definite causal factor in the production of cardiac lesions.

The Inflation of Statistics of Mortality in Diseases of the Heart.

In the New South Wales Official Year Book, 1927-1928,[3] it is stated on pages 316 and 317 that:

> Statistics of mortality in disease of the heart are of limited value, there being important factors connected with the mode of classification which affect the numbers from year to year.

And again:

> The apparent increase in mortality due to diseases of the heart, is probably the result of more specialized biological knowledge and of the greater attention given to pathological diagnosis than to any real cause. Many deaths formerly recorded as being caused by senile decay would now doubtless be assigned to some cardiac trouble.
>
> Eighty-nine per cent. of those who died from heart disease in 1926 were forty-five years or over.

Mention has already been made of the fact that the number certified to as dying of old age is decreasing each year. In 1928, the figures for which have been supplied by the courtesy of the Government Statistician, there is a pronounced decrease, only 784 being tabulated as dying of old age.

This might mean that fewer persons live to the age at which they may be certified to as dying of old age. This is not the case, however, for it is a fact that the average duration of life is increasing

and people are living longer than they did in the past.

That fewer people are certified to as dying of old age is due to the fact, not that they do not live to attain the number of years at which they might be said to die from that reason, but that when certified to as such, if their certificate of death gives a joint cause (primary and secondary) the death is tabulated as being caused by the other disease mentioned. Senility is regarded as an unsatisfactory cause of death and the chances of it surviving in the tabulation of mortality lists is getting less and less. As the end of life is very frequently accompanied by failure of the circulation consequent on degenerative changes in the heart or blood vessels, the association between the two causes is an intimate one and any mention of a cardiac lesion in the certificate of death of an elderly person (unless the death be due to a definite disease such as cancer) will almost surely be tabulated as a death due to heart disease.

It is evident, therefore, that the deaths tabulated under the heading heart disease are inflated to a considerable extent by this means.

The Position of Heart Disease in Mortality Tables When Removed from the Influence of Old Age.

Generally, when the large number of deaths due to heart disease is brought under notice, the influence of old age and the relationship between it and heart disease in statistics is assigned as a reason. While the truth may be admitted in part, it is interesting to analyse the position when this influence is removed.

For this purpose two age periods have been investigated, namely, one from five to forty-four years and one from forty-five to sixty-four years. Table II shows the number of deaths from seven of the principal causes of death during the age

TABLE II.

Showing Deaths in New South Wales from Various Causes of Persons between the Ages of 5 to 44 Years.

Disease.	Year.				
	1924.	1925.	1926.	1927.	1928.
Epidemic, Endemic and Infectious Diseases, Nos. 1–30:					
5 to 44 years	269	237	291	270	290
Under 5 years	383	516	417	492	466
Tuberculosis, Nos. 31–37..	770	685	753	705	772
Cancer, Nos. 43–49 ..	215	199	208	225	207
Pneumonia, Nos. 100–101..	322	272	327	395	337
Puerperal Condition, Nos. 143–150	281	319	268	347	319
Nephritis, Acute and Chronic Nos. 128–129	195	212	212	190	225
Diseases of the Heart, Nos. 87–90	354	359	360	417	398

period five to forty-four years for the years 1924 to 1928. It will be seen that with the single exception of tuberculosis which includes the deaths due to tuberculosis of all systems, heart disease causes more deaths than any of those listed. Table III shows the number of deaths from four principal causes between the ages of forty-five and sixty-four

TABLE III.

Showing Deaths in New South Wales from Certain Causes of Persons between the Ages of 45 and 64 Years.

Cause of Death.	Years.				
	1924.	1925.	1926.	1927.	1928.
Heart Disease	830	931	917	1,130	1,097
Cancer	939	927	890	914	937
Cerebral Hæmorrhage ..	227	324	370	396	326
Respiratory Diseases ..	260	397	432	532	471
Senility	22	18	15	23	14

years. The deaths from senility are included for the sake of contrast only. The progressive increase in the case of heart disease is evident. In the case of both these tables it is necessary to state that the figures quoted should be shown in relation to the number of people living at the ages mentioned. As the years quoted fall in an intercensus period, the information cannot be supplied. It is unlikely, however, that the significance of the tables would be altered in any degree. It will be seen that at every period of life heart disease claims a large proportion of deaths and that the number is progressively increasing.

The information given by Tables II and III allows of a computation of the mortality due to heart disease when the influence of old age is removed.

According to the Government Statistician's report for 1928 the total number of deaths for the whole population in that year between the ages of five and sixty-four was 10,161. For the same year and during the same age period the deaths from heart disease totalled 1,495. Heart disease, then, was responsible for 15% of all deaths during the age period five to sixty-four years. Cancer during the same period in 1928 was responsible for 1,134 deaths, a proportion of 11%.

The Method of Tabulation When Joint Causes of Death Are Assigned in a Death Certificate.

A further reason for the inflation of the number of deaths due to heart disease appears when the system of tabulation where two or more causes of death are assigned comes into operation.

The "International List of Causes of Death" which is in practically world-wide use for the compilation of mortality statistics, deals with the assignment of single terms (diseases), as reported on the certificate of death, to the proper titles of the international list. In other words, for the purpose of being tabulated every death must be attributed to a single cause. In the great majority of cases, however, and for the reason that the form of the certificate seems to require the information, primary and secondary causes of death are furnished. It becomes necessary, therefore, to provide for the consistent and uniform distribution of such deaths in order that statistics, when compiled in the usually accepted manner on the basis of a single cause for each death, shall be comparable.

The desirability of uniform treatment of joint causes of death has long been recognized. In fact if the trend of mortality from various causes is to be accurately measured year after year, the uniform treatment of joint causes of death is not only desirable, but necessary, if the death of each person is to be tabulated as though due to a single cause.

In 1914 the Bureau of Census in Washington published an "Index of Joint Causes of Death." It did not cover all the combinations of joint causes which appear on certificates, so that the tabulator who tried to follow the index often failed to find the combination of causes for which he was searching and so was obliged to make the decision as best he could. Sometimes it was possible to reason out a decision from some other combination of causes regarded as analogous, but here again different tabulators would not happen to pick out the same combinations as analogous, so that considerable differences in the classification of causes of death have been inevitable, even when different registration officers have tried to use as a guide the 1914 "Index of Joint Causes."

The treatment of joint causes has never been adequately discussed by the international conferences nor by any body, so this "Revised Manual of Joint Causes," 1925, is published as a temporary guide for those who are groping for help in making their assignments rather than as an authoritative manual.

It is regrettable that the existence of two or more causes of death cannot be shown on account of the extra expense of tabulation and printing.

The tabulation of causes of death shows the assignments preferred by the Bureau of Census and permits of more accurate interpretations.

This manual comes from the Department of Commerce, Washington, United States of America, and incidentally the present President of the United States, Herbert Hoover, was Secretary of that Department in 1925, when the latest edition was published. It is at present in use in the State of New South Wales and has been for some time. Those responsible for its production recognize that it is open to criticism, when they state it is too much to expect all physicians and registrars to agree with the relative importance or weight shown for certain combinations in this manual. Different points of view regarding the possible sequence of joint causes may be responsible for difference of opinion. No two individuals would probably ever agree regarding preferences in so many thousands of combinations. The decisions in the "Manual of Joint Causes of Death" relate to bare statements of causes of death without consideration of duration, place of death, age, occupation or any such expressions as "following on," resulting from," "due to" *et cetera*.

The Results of the System of Tabulation.

The results of this system are evidenced more particularly and exert their greatest influence in the case of deaths attributed to heart disease and supply a further explanation of the large number of deaths under that heading. At the same time whatever criticism may be levelled against the system, we as medical practitioners cannot escape some of the responsibility for the results. The form of death certificate used requests the duration of the causes of death to be stated. Admittedly such information may be difficult to give with accuracy in some cases, but according to the Government Statistician in 70% of cases no duration times whatever are given. While the tabulator may exercise his judgement in certain cases when allocating the cause of death, his task is rendered much more difficult if he is not supplied with the necessary data to guide him. For the sake of uniformity he must refer to the manual and give preference according to the instructions.

A few examples will best illustrate how the system of tabulation operates. These are taken at random from a number of death certificates issued in this State during the first quarter of 1929 (see Table IV).

TABLE IV.

Age at Death in Years.	Primary Cause.	Duration.	Secondary Cause.	Duration.	Tabulated for Statistics.
50	Pernicious anæmia	5 months	Endocarditis	5 months	Endocarditis
64	Pernicious anæmia	Not stated	Myocarditis	Not stated	Myocarditis
48	Uræmia	"	Myocarditis	"	Myocarditis
59	Broncho-pneumonia	12 days	Fatty degeneration of heart	"	Myocarditis
43	Exhaustion of acute mania	21 days	Acute myocarditis	"	Myocarditis
65	Cholelithiasis	Not stated	Chronic myocarditis	"	Myocarditis
52	Myocarditis	"	Peripheral neuritis	"	Myocarditis
45	Acute colitis	"	Myocarditis	6 days	Myocarditis
71	Intestinal obstruction	"	Chronic myocarditis	Not stated	Myocarditis
72	Enlargement of prostate, uræmia	5 years	Chronic vascular degeneration	"	Myocarditis
43	Cholelithiasis	Not stated	Myocarditis	"	Myocarditis
43	Acute intestinal toxæmia	14 days	Myocarditis	"	Acute myocarditis
82	Acute cholecystitis	Not stated	Cardio-vascular degeneration	"	Myocarditis
60	Acute parenchymatous nephritis	5 months	Myocardial degeneration	"	Myocarditis
58	Erysipelas	4 days	Myocarditis	"	Myocarditis
60	Acute catarrhal jaundice	Not stated	Myocarditis	"	Myocarditis
47	Broncho-pneumonia	4 days	Myocarditis	"	Myocarditis
40	Intestinal obstruction	Not stated	Myocardial degeneration	"	Myocarditis
59	Influenza	4 days	Myocarditis	"	Myocarditis
71	Gastro-enteritis	Not stated	Myocarditis	"	Myocarditis

A perusal of the list will show the regularity with which myocarditis is assigned as a secondary cause of death. Its regularity is as constant as the primary cause is varied. The effect on statistics is indicated by the tabulation which is here presented. There is no doubt that, leaving heart disease out of the question, statistics are vitiated. Pernicious

anæmia would certainly seem to warrant preference, no matter what company it was in.

To what extent the system of tabulation is affecting the total figures in the case of heart disease only a rough calculation may be made. For the year 1928 a liberal estimate would be 500. This number, subtracted from the total number for that year, namely 3,849, leaves 3,349 and the position of heart disease is not materially affected.

While one hesitates to offer criticism on a subject which has evidently many intricacies and bearing in mind the remarks quoted at the outset that the existing classification has been developed by men who were both medical practitioners of high standing and statisticians of eminence, it still appears that the intention of the practitioner signing the certificate loses its expression after it has been through the hands of the tabulator.

To find that a certificate giving pernicious anæmia or acute cholecystitis as a primary cause of death is attributed in statistics to heart disease, if myocarditis be given as the secondary cause, will come as a surprise to many practitioners.

Those who are responsible for the tabulation of causes of death in this State, are fully seized with the difficulties presented by the many combinations of joint causes and are assiduous in their efforts to obtain uniformity. The manual issued by the Bureau of Census in Washington was adopted for this purpose. The late Sir George Knibbs (*loco citato*) in the article referred to above states that "every revision of the classification, advances in science generally and in medicine, indicate the need of some change." Space will not permit to narrate the remedies suggested by him. The next revision by the International Conference takes place in 1930 and one important recommendation of his is expressed as follows:

> Experience has shown that the compiler in a statistical bureau who daily sees the type of citations of cases by the practising doctors of a country, gets to know something of the limitations of their certifications, and also whether all the diseases of the territory for which he compiles appear in the classified list. For this reason it is desirable that, say, the Commonwealth Statistician should be represented on the local revision committee and that committee should be appointed without delay and at once get in touch with the Secretary-General of the International Committee on the other side of the world.

One suggestion of importance given in America would materially assist the tabulator, namely, that "if the causes are unrelated, one not being the result or complication of the other," the disease most important as the cause of death should be entered first without regard to relative duration, the word secondary being struck out on the form.

With regard to the question of the frequency of the certification of myocarditis as a secondary cause of death, such frequency implies that it is a common affection. In 1928 it was responsible for 63% of the deaths due to heart disease. There are no figures prior to 1928 which will give the relative frequency of valvular disease as a cause of death, but in that year the percentage was 20.

It may be allowed that there is every justification for the certification of the secondary cause as myocarditis and it is not to be understood from anything that may be said in this connexion, that the diagnosis is questioned.

It is fair to assume, however, that it is very frequently a terminal event and when it is judged to occur as such it is especially asked (see note to 88 and 90 in "International List of Causes of Death" in book of certificate forms as issued to practitioners in this State) that it be not reported. Observance of this request will do much to obviate what is happening at present and enable the figures with regard to heart disease to be presented with greater accuracy and incidentally and perhaps of equal importance to give the proper significance to other diseases which may now be escaping under the present system their tabulation in mortality lists. (Compare the association of pernicious anæmia and myocarditis in the list of certificates in Table IV.)

Myocarditis as a diagnosis may be said to be "having a run." As a matter of fact its recognition both *intra vitam* and *post mortem* is often a matter of great difficulty and its presence is often assumed. As Holst[4] states: "It does not signify diagnostic progress; it is rather to be considered as diagnostic resignation."

Instances of similar faults in the certification of causes of death are supplied in the case of uræmia and malignant disease. Practitioners are specially requested (see "International List of Causes of Death," VII) never to report mere names of symptoms as uræmia. With regard to malignant disease it is requested that the nature of the tumour should be stated when possible.

The Significance of the Increased Mortality from Heart Disease.

If it be admitted that the number of people dying from heart disease is increasing and the available information warrants the conclusion that it is, it would also seem to be true that there is some complacency about the fact.

Some reasons for this suggest themselves. Heart disease exerts its greatest influence in mortality statistics after the age of sixty-five years, though as has been pointed out, it is still potent before that.

The same is true of cancer, but the comparative comfort of the one compared with the dread of mind and distressing accompaniments of the other make a world of difference so far as our attitude towards the two is concerned. Heart disease may exist for years and call for little more than limitation of activity to the sufferer and as a means of terminating life it may be the most merciful and entail the least suffering of any disease. Death is an inherent attribute of life and someone has said we are all necessarily walking to our own funerals. But, if the journey be not attended with suffering and its termination still indefinite (or we be pleased to think so) it does not cause anxiety. The decrease in the number of deaths due to old age and their

transference to the column of heart disease is, after all, only changing the name. The connexion is expressed in the old adage, "a man is as old as his arteries," and no doubt the great majority of old people dies from old age of the arteries. Vascular tissue throughout the whole of life is called on to take a large share in the process of life and bear the strains thereof. What should concern us, then, in the particular case of heart disease, is that the age at which our arteries grow old or show the changes associated with old age, may be decreasing and in this regard the stress of modern life may be playing an important part. Further investigation is necessary to determine whether the age incidence of heart disease is altering in the sense that more people are dying from it now than hitherto at the same ages.

The Incidence of Heart Disease.

Owing to insufficiency of data it is not possible to make any estimate of the incidence of heart disease. It has no urgent needs and from a public health point of view presents no difficulties as scarlet fever, diphtheria or tuberculosis. Whether there is any degree of association between social or economic status and heart disease is also difficult to determine in the absence of data.

It is doubtful, yet we may be blind to the fact, whether we have reached the stage in this State that has been reached in the State of New York where the economic aspect of heart disease has compelled its recognition as a community problem and prompted an inquiry which was suggested by the large and increasing number of deaths from heart disease, as well as the large number of children in the public schools of New York City who were afflicted.

Haven Emerson,[6] of New York, in an address to the American physicians at Washington on May 2, 1928, said:

> No small part of the present colossal wealth of our country is due to the accumulated savings of the lives of men and women in the years of their greatest material productivity by the fall in the death rate from pulmonary tuberculosis. Now that tuberculosis has lost its leadership of the column of deaths and heart diseases have emerged, partly by the fall in death rates from other causes and partly because of the great and sustained increase in the death rates from heart diseases, popular interest, as well as medical, drives us to analyse the causes and costs in this field of pathology. No better confirmation of the adage, "The first wealth is health" is to be found than the estimate of economists that the value of human lives in this country is at least five times the value of all material possessions.

That the position in this State with regard to heart disease differs from that in others where the rates are high, may be a matter for speculation, but only for reasons that are themselves speculative. In other words, the rates of mortality being, as has been shown, not accurate so far as heart disease is concerned, they are nevertheless the only means at hand for making an estimate and at least call for analysis.

Taking all the known factors into consideration, however, and giving them due weight so far as possible, it is difficult to escape the conclusion that heart disease is on the increase.

Causes of the Increase.

As was said at the outset, the primary object of these observations was to find a cause for the increase in the deaths attributed to heart disease. Certain factors operating to swell the numbers have been discussed.

Apart from these the causes are not easy to assess. The ætiology of heart disease is diverse, the field of speculation correspondingly wide. Explanations, when offered, are vague and though plausible enough, have little scientific value. For example, such terms as "the speed at which we live," "the wear and tear of modern life," "intestinal intoxication" are all put forward as causes, but no facts are elicited to support the contention that they are truly causative.

One accompaniment of modern life there is, however, of increasing frequency which, whether recognized or not by the individual, is a definite causative factor in heart disease. It is suggested that it is only more frequent since the introduction of the ready instrumental means of measuring it. This may be so, but it does not alter the fact that under the familiar name of "blood pressure" hypertension or hyperpiesis, as it has been called by Clifford Allbutt, is a definite disease and is now recognized as such. It indicates that certain pathological involvements accompany or are entailed by the increased blood pressure. There are no means of estimating its incidence in this State; consequently expressions of opinion on the question can be given as personal and therefore of relative value only. Mosenthal[5] states that a study of mortality statistics readily demonstrates that hypertension is one of the most common causes of death, probably being of equal importance to cancer and tuberculosis.

It finds no place in mortality lists in this State and while not suggesting that it should, there is no doubt that it holds a prominent place as a true primary cause of heart disease in those over forty-five years of age. It is true that we are ignorant of its cause, though all the "transferable tickets" (Clifford Allbutt) such as "wear and tear" *et cetera* are put forward to explain it.

Conclusion.

It has been shown that the information to be gained from mortality tables in this State in regard to heart disease is to be accepted with reserve on account of various factors.

Only efficiently directed study of the problem will elucidate the true facts. That it warrants investigation is undoubted on the figures.

There are no existing means of estimating the incidence of heart disease in the population.

From the data available the conclusion follows that heart disease is on the increase in this State

and is responsible for a large and increasing death rate.

So far as these observations have gone, the claim of heart disease to the title, "Captain of the Men of Death," may not be disputed.

Acknowledgement.

The tables and various statistics contained in this article have been obtained from the Bureau of Statistics, Sydney, New South Wales.

My thanks are due to the officers of that department for their unfailing courtesy and valuable help in the compilation and checking of the figures.

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Reviews.

THE PROBLEMS OF OBSTETRICS.

SERIOUS attention is demanded of a work reflecting such a wide obstetric experience as that of Dr. Henry Jellett, formerly Master of the Rotunda and Professor of Midwifery at the University of Dublin, whose latest book on "The Causes and Prevention of Maternal Mortality" makes a special appeal by reason of the thoroughly practical way in which he puts that experience at the service of a task admittedly difficult.[1]

In his introductory chapter Dr. Jellett writes: "We make medical practitioners and midwives, but we fail to provide them with a sufficient knowledge of midwifery." In two chapters on the education of the maternity nurse and midwife and the education of the medical adviser he deals fully with their requirements. As Consulting Obstetrician to the Department of Health, New Zealand, he has first hand evidence of the value of the course for midwives and maternity nurses prescribed by the Nurses and Midwives Registration Board for New Zealand, which he gives in detail, comparing it critically with the curricula of the Central Midwives Board of England and Wales, Denmark and Holland. In contrasting the low maternal mortality rate of Holland and Denmark with the comparatively high figures for New Zealand and England and Wales, he draws attention to the much longer periods of training for the midwife in Holland and Scandinavia, where almost all normal labours are conducted by midwives, and further points out that "the obstetrical knowledge or at any rate the opportunity of obtaining it, of the average medical practitioner in Holland and the Scandinavian countries is greater than in the British Empire and skilled specialists are more readily available to come to his assistance."

Dr. Jellett expresses himself in no uncertain fashion. He will find every experienced practitioner in agreement with his "doubt of the wisdom of trying to train every maternity nurse to be a midwife" and he defines clearly the training and the rôle of each in the obstetric scheme, a valuable contribution.

His chapter on the education of the medical adviser is a well reasoned appeal for more intensive and comprehensive training in obstetrics: "It is quite correct to say that 80% or 90% of women will deliver themselves without assistance. So they will if they are allowed to do so. This is where the half-educated practitioner fails, for he does not realize that it is necessary to allow the labour to be normal."

In analysing the causes and prevention of maternal mortality Dr. Jellett speaks of "personal" and "impersonal" causes in a new sense, the meaning of which he is at some pains to make clear, his opinion being that "all the most common causes of maternal mortality (on the official list) are or can be made 'personal' causes and as such can be removed." These are sepsis, eclampsia and the allied toxæmias of pregnancy and labour and almost all deaths from the accidents of labour. While we cannot quite follow Dr. Jellett to the border-line which separates the "personal" from the "impersonal" in this regard, we are wholeheartedly with him in his insistence on the fact that "unpreparedness" is more often than not the cause of failure to save life.

Dr. Jellett has ranged over a wide field of inquiry. He writes from a sense of conviction and has the courage of his opinions. He quotes appositely and with appreciation from the reports of Dame Janet Campbell, Professor Marshall Allan, of the University of Melbourne, and Dr. Sydney Morris, of the Department of Public Health of New South Wales. His book is helpful, suggestive, interesting. It should be not only educational to the student and midwife, but stimulating also to those who are responsible for their training.

[1] "The Causes and Prevention of Maternal Mortality," by Henry Jellett, B.A., M.D. (Dublin), F.R.C.P.I.; 1929. London: J. and A. Churchill. Demy 8vo., pp. 307. Price: 15s. net.

RATS.

A. MOORE HOGARTH who was largely responsible for the introduction of the Rat Destruction Bill which was "laughed out" in the House of Commons in 1908, but which became law in 1919, summarizes in "The Rat, a World Menace," the history, geographical distribution, the habits, the economic loss and the best methods of combating the pest.[1] In the attempt to condense the vast storehouse of information in such standard works as those of Boelter and Hovell the author has sacrificed much that is valuable to the man actually engaged in catching rats.

The tables supplied by the Ministry of Agriculture (England) to distinguish the black and brown species, by the Institute for Medical Research, Kuala Lumpur, on the human diseases spread by rats, the extracts from Dr. Andrew Balfour's "Observations on Wild Rats in England" and the prescriptions of rat poisons issued by the Ministry of Agriculture are the most valuable features of the book.

With regard to the Australian Rodier system of extermination by killing the females and liberating the males, the author by his optimistic review endorses the scientific basis of the idea, but does not sufficiently emphasize the impractibility of applying the method in a large city.

Experience in Sydney which covers nine outbreaks of plague since 1900 and includes the slaughter of over a quarter of a million rats in the last outbreak in 1922-1923, showed that phosphorus is the most satisfactory poison. Barium carbonate and squills which have of recent years been so highly lauded on account of the lessened domestic risk, have not proved so effective as would be expected from the eulogism of their advocates.

[1] "The Rat, a World Menace," by A. Moore Hogarth, F.E.S.; 1929. London: John Bale, Sons and Danielsson, Limited. Pp. 112. Price: 7s. 6d.

The Medical Journal of Australia

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A Retrospect.

Oto-Rhino-Laryngology.

THERE have been several useful controversies and discussions on subjects coming within the scope of otology, rhinology and laryngology during the year 1929. Much has been written on the general subject of deafness. While no new suggestions have been advanced in regard to relief measures, many otologists claim that treatment of various nasal and faucial conditions in the early stages of deafness results in the arrest of the process. The subject of oto-sclerosis has engaged the attention of many workers. O. Mayer has evolved an interesting hypothesis of the pathology after a prolonged study. He postulates that the oto-sclerotic areas are pathological growths caused by congenital malformations; he denied all inflammatory processes in the causation. Brunner teaches that oto-sclerosis is a dystrophy depending on an unknown metabolic disturbance in the bone and chondroid tissue supporting the labyrinth. Döderlein has examined the temporal bones after death and has found that the primary changes in oto-sclerosis occur near the blood vessels. These changes resemble the changes encountered in osteomalacia and rhachitis. In spite of the careful investigation of many specimens, he has not been able to discover any changes earlier than the fourth year of life. He claims that this indicated a post-embryonic commencement.

J. N. Watt is of opinion that the main cause of deafness is a lack of aeration of the middle ear through the Eustachian tube. He has therefore dealt with the condition by opening the mastoid antrum through the usual mastoid incision and exposing the *aditus*. A gold tube is fitted from the surface of the *aditus*. He claims good results from this operation. Ness advocates a permanent intra-meatal antrostomy for this form of deafness. Woods employs intratympanic injection of iodine in the treatment of early oto-sclerosis. Dan McKenzie injects massive doses of "Collosol Iodine" intravenously. He has formed the opinion that this treatment benefits some patients. Some of the speakers at a meeting of the otological section of the Royal Society of Medicine claimed that improvement in hearing follows the application of so-called medical diathermy. No indications were given of the type of patient who might benefit by this treatment.

H. B. Shaheen states that good results can be obtained in suppuration of the middle ear and of the mastoid antrum by the subcutaneous injection of boiled milk. In regard to the operations for mastoid disease many modifications have been suggested. Harold Kisch has advocated the insertion of a tongue of the temporal muscle into the mastoid cavity after the operation; the wound is sutured over the muscular pad, but no meatal flaps are used. He is of opinion that healing takes place more rapidly and that the post-operative manipulations are painless. R. Godsall has expressed the view that the radical operation yields better results if no flaps are cut and if the post-operative treatment is carried out through the posterior wound. J. Lempert recommends a transmeatal route; he performs antrostomy with the aid of an engine-driven burr.

Szenita has observed tuberculous *otitis media* in patients in sanatoria. He has reason to believe that in children the infection passes to the middle ear by the blood stream, while in adults it spreads up the Eustachian tube. He has seen very few complications arising from tuberculous disease of the middle ear.

Meningitis occurring as a complication of *otitis media* is known to be a very fatal condition. Jenkins claims that recovery may be gained by washing out the subarachnoid space and by taking steps to maintain a normal pressure of the cerebro-spinal fluid while the mengineal tissues are being exposed. Queckenstedt's sign is sought in sinus thrombosis as a regular diagnostic measure both in Germany and in America. When the sinus is obstructed, pressure on the jugular vein does not alter the pressure of the cerebro-spinal fluid.

Improved X ray technique enables otologists to detect even small variations in anatomical formations in deafness. It is becoming an essential method of examination. Krummel has described a new method of examining the mastoid processes by means of X rays. The skiagrams are taken in an antero-posterior position and both processes are shown on one negative. Full details are seen on both sides simultaneously.

Mosso has carried out some highly interesting experiments to determine the absorption of drugs from the external auditory canal. Opium, cocaine and carbolic acid give rise to hyposensibility of the vestibule. Potassium salts introduced into the external auditory canal are excreted in the saliva, passing through the lymphatic vessels.

K. Grahe has called attention to the fact that the vestibular reactions are not altered in the human subject after removal of a large part of the cerebellum. The significance of this finding is not clear. Otherwise no new facts have been elicited in regard to labyrinthine function. Larsell and Fenton have studied the embryology and neurohistology of the spheno-palatine ganglion and its changes in pathological conditions.

The adrenalin probe reaction, devised by Much, has been further studied by Joöl. The turbinate is sprayed with adrenalin solution; when the turbinate is stroked with a probe in normal individuals a red line appears; in the presence of certain constitutional disabilities a white line is seen instead. The reaction has been noted in syphilis, tuberculosis and some nervous diseases and also during pregnancy.

Little advance has been made in rhinology. The subject of ozœna continues to tax the powers of the clinician. Some suggestions in regard to methods of treatment have been made, but no fresh facts concerning its nature have been elicited. Grave and Pickworth have undertaken a useful investigation of the condition of the nasal sinuses in patients in a hospital for the insane. They offer evidence in favour of the view that septic foci at times act as contributory causes of mental disease.

The laryngologist appears to be claiming the œsophagus as well as the pharynx and larynx as his domain. Radium and radon seeds are being used more freely in malignant disease of the œsophagus. These agents have proved highly disappointing in cancer of the larynx and of the post-cricoid area. For laryngeal cancer St. Clair Thomson claims permanent cures in approximately three-quarters of patients who have been subjected to laryngofissure.

Paresis of the larynx as a sequela to enteric fever has been described by Roy, while Mollison has encountered paresis of the pharyngeal muscles leading to dysphagia.

The diagnostic and therapeutic use of "Lipiodol" for pathological conditions of the bronchi and lungs continues to find favour. The injections are made by some laryngologists through the bronchoscope, while others prefer to use a syringe.

A great deal has been written on the treatment of diseased tonsils. Enucleation appears to be the favourite method in Europe, although diathermy has many friends. Of the diathermic methods the *morceleur* and the snare are preferred to the monopolar and the bipolar methods. It is generally held that patients should be kept in hospital for at least forty-eight hours after removal of tonsils and adenoid vegetations. J. I. Thomson has described a condition known as agranulocytic angina and appeals to other surgeons to report any cases observed. The condition is associated with severe anæmia and is often fatal. It appears that staphylococci and streptococci are found in the smears.

Neurology and Psychiatry.

Among the most important events of the year in the realms of neurology and psychiatry is the opening in Edinburgh of the Jordanburn Nerve Hospital and Psychological Institute. The organization embodies departments for organic nervous diseases, psychiatry, mental deficiency, research in psychology, child guidance and social service. The provision of machinery for all this team work under one roof is a novel, even unique departure and is significant of the trend of thought in this direction. In recent years a tendency has become manifest to hand the problems of mental deficiency, abnormal mental reactions and the like to non-medical

psychologists. These problems are medical and cannot be satisfactorily solved unless the pathological changes of all the organs and tissues are coordinated with the abnormal physiological responses. It is therefore eminently desirable to coordinate all the departments named above under one scheme and to subject all nervous, psychological and psychiatric phenomena in terms of physiological response.

Epidemic encephalitis has been discussed anew and attention has been paid to post-encephalitic phenomena. It has been demonstrated that among other sequelæ convulsive disorders, changes in the ocular fundi, various forms of muscular atrophy and certain peculiar derangements are commoner than was previously thought. Jelliffe has undertaken a particularly complete review of all that has been written on those complicated phenomena arising in Parkinsonians and known as oculo-gyric crises. His review comprises a dissection of hundreds of published records. He favours the view that the disorder is a compulsion neurosis.

In addition interest has been focused on forms of encephalitis and myelitis consequent upon vaccination, morbilli, exposure to certain trade chemicals, such as benzene, the use of certain therapeutic sera and some of the arsenical preparations. It is evident that the neurologist must be aware of the changes that take place in such circumstances, in order that he may deal intelligently with them when the complications arise.

In the treatment of general paralysis of the insane inoculation with malarial blood or plasmodium continues to hold its usefulness. It is true that the accuracy of the diagnosis of general paralysis of the insane under present day conditions has been questioned and in consequence the validity of the figures bearing on the subject has been challenged. Support for the treatment has been provided by histo-pathologists who have claimed that spirochætes can no longer be found in the brains of general paralytics who have been infected with malaria. The corollary is that the plasmodium of malaria must be truly spirochæticidal. A point for observance in practice has been emphasized that treatment should be continued as long as a serological reaction is obtainable.

Notwithstanding that in some quarters the usefulness of operations on the sympathetic nervous system devised by Royle has been doubted, the results obtained by him and others who have faithfully repeated the technique prescribed, are too cogent to be set aside. Wilkinson has demonstrated very clearly that voluntary muscle fibres are innervated solely by somatic nerves, that is, cerebrospinal nerves. The sympathetic nerve supply of striated muscle tissue is distributed to the blood vessels and is said to regulate the circulation in the muscles. It is suggested that the alteration in muscle tone after ramisection is referable to vasomotor changes and not as was suggested by Royle and Hunter to the elimination of a sympathetic nerve supply of muscle fibres, which according to Hunter's hypothesis maintained muscle tonus.

The subject of cerebral tumours was discussed at the Neurological Congress in Paris and has also occupied considerable space in literature. Records have now become available of every kind of tumour arising in every conceivable situation. By this means the diagnosis of cerebral tumours has been greatly facilitated. Physical methods of localization, including Dandy's ventriculography, have been further tested and seem to be regarded as established procedures. In regard to treatment surgical measures are being improved; for inoperable tumours deep X ray therapy is gaining more advocates.

The Melbourne school of medicine is to be congratulated on advancing knowledge of acute anterior poliomyelitis and in particular in providing incontestable proof of the virucidal action of the serum of convalescents, as well as in emphasizing that there are signs by which the disease can be diagnosed in the preparalytic stage when the injection of serum is likely to do good.

Among other subjects on the symptomatology or pathology of which instructive contributions have appeared, mention may be made of head injuries, increased intracranial tension, post-traumatic neuroses, epilepsy, *tabes dorsalis,* muscular dystonia, *myasthenia gravis, dementia præcox,* mongolism and psychoses following influenza and pernicious anæmia.

A useful discussion took place on the value of the psycho-galvanometer at the Australasian Medical Congress (British Medical Association), Sydney, 1929. The place of the psycho-galvanometer and the significance of the reactions have not yet been exactly defined, but evidence is available of its value in the measurement of the intensity of emotional states.

Morbid Anatomy.

The past twelve months have been a time of steady advance in morbid anatomy. There have been no striking discoveries, but some new ground has been broken and ground previously won has been explored and used. This is most noticeable in connexion with the work of Kolodny in the Register of Bone Sarcomata, undertaken for the American College of Surgeons. Kolodny's classification has been widely adopted and used in the literature during the last year. The view has been expressed that similar work should be undertaken in other spheres and there is no doubt that this would be attended by good results.

In an attempt to review recent work attention naturally turns first of all to malignant disease. Further investigations have been carried out on the cytology of malignant tumours. Those of Lumsden and Kohn Speir and of Pybus and White have been discussed in these pages. Though much of the work is highly interesting, it is so far impossible for the cytologist to differentiate the serum of an animal with malignant disease from that of an animal not so affected. Baker, Dickens and Gallimore have found a great difference between the normal tissues and malignant tumours as far as the anaerobic metabolism of glucose is concerned. The tumour tissues produce from five to ten times as much lactic acid as the normal tissues. Gruskin has advanced the hypothesis that each type of normal cell produces a lysin against other types of cell. If this lysin is not produced, cancer results when the proper extrinsic factors are present. He believed that a lytic agent might be produced by the inoculation of animals with purely embryonic epithelial cells in the presence of carcinoma and connective tissue cells in sarcoma. He has elaborated a test and has used it for diagnostic purposes. He claims that the results demonstrate the value of the test as a clinical measure. Cherry has continued his work on tuberculosis and malignant disease and reported some further results at the third session of the Australasian Medical Congress (British Medical Association). Sampson Handley has suggested that tuberculous infection leaves behind some lymphatic obstruction and that this obstruction prepares the soil for the subsequent development of malignant changes. He holds that lymph stasis plays an important part in the ætiology of malignant disease. There are signs that the group of tumours frequently described as endotheliomata are receiving more critical consideration. Ewing has drawn attention to the necessity for careful discrimination. In other words, there is a tendency to revert to the doctrines of Ribbert. Sargent and Greenfield have investigated the hæmangiomatous cysts of the cerebellum. They find that most cerebellar cysts have capillary hæmangiomata in their walls. They find a close relationship to trauma of the head in certain instances and suggest that trauma may start the formation of a cyst in a patient who already has an angioma of the cerebellum.

André Jousset has carried out chemical and histological investigations and claims that anthracosis is nearly always a siderosis. He finds that the pigment is an albuminous compound of iron. He believes that all the organs and especially the lungs can under inflammatory stress acquire the normal function of the liver, a capacity of mobilizing the waste iron pigments.

Mackey and Hamilton Fairley have continued their work on sprue. As a result of the study of *post mortem* specimens they conclude that there are probably two distinct factors in the production of the sprue syndrome, a specific virus acting on the intestinal tract, resulting in degeneration, atrophy and diminished resistance of the mucosa to bacterial invaders and secondary bacterial invaders and their toxins causing toxæmia and acting on the cells of the parenchyma of the viscera and of the erythroblastic tissue of the bone marrow.

As a result of experimental work Coryllos and Birnbaum have advanced the view that lobar pneumonia is primarily an atelectatic condition. This

conception offers an explanation for the lobar nature of the disease and of the radiographic appearances.

A. J. Trinca has investigated fat necrosis of the breast. He finds that the condition is not necessarily an independent lesion in the breast and that trauma is not an essential ætiological factor.

McGregor and Simpson have reported investigations into *thrombo-angiitis obliterans* and have found histological evidence to support the view held by Buerger that the condition begins as an acute inflammation. Buerger has also found that paravascular implantation of clot from patients with *thrombo-angiitis obliterans* is followed by typical lesions in apparently healthy veins of inoculated persons.

Hutton and Young in Scotland and Newland and Woollard in Australia have focused attention again on chordoma and have emphasized its notochordal origin.

Physiology.

Among the more important investigations into physiological problems recorded in 1929, the demonstration by W. B. Cannon and his fellow workers is perhaps the most striking. They demonstrated that small animals can live and maintain apparently normal health in the complete absence of the sympathetic nervous system. Animals whose sympathetic chains have been removed on both sides, performed their ordinary functions for many months, even including the production and care of their young. The experiments yield a further demonstration that cats can live for months without a detectable amount of adrenalin passing into the circulation. The only obvious difference between these animals and normal cats is that after removal of the sympathetic system the animals are much more sensitive to cold temperatures. The conclusion drawn is that the sympathetic system is dispensable in the protected conditions of the laboratory, but that it finds its more important service at times of cortical emergencies.

Hugh MacLean and others working under his direction have clarified ideas concerning gastric acidity and the changes after a test meal. They have produced evidence to the effect that there is an automatic regulation of the gastric acidity. The presence of a certain concentration of acid inhibits the secretion of acid with the result that a neutral fluid containing chlorides is secreted and when this is diluted, it reduces still further the acidity of the gastric contents. They found that if regurgitation occurs from the duodenum to the stomach, it is small in amount and of no importance from the point of view of lessening the acidity of the gastric contents. They proved further that the peptic activity of the stomach contents rises *pari passu* with the increase of neutral chlorides which make their appearance with a diminishing acidity.

R. S. Aitken maintains that the renal threshold for chlorides is not a definite point and suggests that the conception of a renal threshold for chlorides be abandoned.

J. Mellanby has isolated a substance which he believes to be secretin. It is a polypeptide containing phosphorus. One part in five million parts of blood produces a normal secretion of pancreatic juices. Swale Vincent and J. H. Thompson claim that the adrenal cortex secretes a substance, called by them pneumin, essential for the normal movements of respiration. Others deny that there is any such substance. Kamm and others working with him have separated almost completely by fractional precipitation the oxytocic and the pressor principles of the secretion of the posterior lobe of the pituitary body. Preparations containing these two principles are now on the market under the names of "Oxytocin" and "Vasopressin." Draper claims that the renal activity of renal extracts is due to a third substance.

The grave difficulties associated with the accurate determination of the cardiac output have impelled investigators to search for simpler and better methods than have hitherto been employed. A. Grollman has endeavoured to employ acetylene as an inert gas for this purpose. He claims that he has obtained good results. Moore, Kinsman, Hamilton and Spurling have injected a substance, sodium phenol-tetroid-phthalein of Mallinkrodt, which they find to be easily detectable. The injections are made into a vein and consecutive samples of blood are taken from an artery. The cardiac output per minute can be calculated from the ascertained concentration of the dye in the various samples.

Abstracts from Current Medical Literature.

PÆDIATRICS.

Lobar Pneumonia.

IT is a matter of dispute as to whether alveolar (lobar) pneumonia occurs throughout childhood and is fairly common in infancy. C. McNeil, A. R. MacGregor and W. A. Alexander (*Archives of Disease in Childhood*, April, 1929) report a series of twenty-three fatal cases of alveolar pneumonia, the majority of them in children under two years, in which the *post mortem* evidence justifies the diagnosis of this type of pneumonia. The ages of the children ranged from four months to four years and nine months, more than 75% being under two years of age. There were seventeen boys and six girls. In fifteen the pneumonia was complicated as follows: Empyema in eight, pericarditis in three, meningitis in three, abscess of lung in three, peritonitis in one, acute nephritis in one, suppurative pyelonephritis in one, congenital defect of the heart in one. The site and extent of the consolidation varied considerably, far more than in the corresponding disease of adult life. It was usual to find only a part of a lobe or lobes affected. The main problem for the pathologist is to distinguish between alveolar (lobar) pneumonia and confluent bronchopneumonia. The affected part in the former is massively and completely consolidated and, as every alveolus is filled with exudate, no areas of collapse are present. It lacks the nodular character and somewhat rubber-like resilience which in most bronchopneumonic lungs, even of the most confluent type, are produced by alternating zones of consolidation and of partial collapse. A cut section has a dry, dull, finely granular appearance, due to the projection of plugs of exudate from the alveoli which contrasts with the moist and shiny surface of a section through bronchopneumonic lung. The bronchi and bronchioles are not conspicuous, their walls have no inflammatory thickening, are not noticeably dilated and do not contain large quantities of pus, although a certain amount of exudate is present in them. The absence of bronchitis, especially of the smaller tubes, in those parts in which there is no consolidation, is an important feature in typical alveolar pneumonia. The tracheo-bronchial and root glands do not become nearly so inflamed in the alveolar type as they do in broncho-pneumonia. Histologically it is characteristic of alveolar pneumonia that the products of inflammation are thrown out into the alveolar spaces and that the walls do not manifest any great degree of infiltration or inflammatory change. Though the bronchi in the pneumonic area are usually filled with exudate similar to that in the alveoli, evidence of severe inflammation of their walls is absent. This freedom of the bronchial and alveolar walls and stroma generally from damage by infiltration with inflammatory cells is characteristic of alveolar pneumonia as opposed to broncho-pneumonia. The author gives details of three illustrative cases.

Tuberculous Infections in Households.

A. I. BLAU (*Archives of Pediatrics*, April, 1929) has made a study of the influence of active tuberculosis in the household upon the development of tuberculous meningitis in children. The study embraces 110 patients with proved tuberculous meningitis. Of these 25% gave a definite history of exposure to active tuberculosis in the family. The author points out that, since meningitis is only one of the manifestations of the disease, the chances of infection must be very great. Two-thirds of the patients in whom exposure could be demonstrated, had a father or mother or both with active pulmonary tuberculosis. Prolonged exposure was not necessary for the development of infection. While in most instances the exposure lasted several months, in one it was only for two days, and in another eleven days. The disease did not manifest itself in less than four months after exposure and in some instances not until a year or more afterwards. The development of the disease was often brought about by some debilitating influence or some intercurrent disease such as measles, pneumonia, pertussis or influenza. In 3% of the series a definite family history of tuberculosis was obtained, but no history of contact. The author considers that prompt removal of a tuberculous individual from contact with children warrants serious consideration by health authorities.

Intraperitoneal Injections of Blood.

W. C. C. COLE AND J. C. MONTGOMERY (*American Journal of Diseases of Children*, March, 1929) present their results after giving 237 blood transfusions to children by the intraperitoneal method. Although it is possible that blood grouping is not necessary, this precaution was not omitted. The blood was in every case carefully citrated and was run by gravity into the peritoneal cavity. The peritoneal needle was inserted in the mid-line just below the umbilicus and was of large bore and short bevel type. About 120 cubic centimetres (four ounces) was the usual amount given to children under three years of age and it was found that the therapeutic effect of repeated small transfusions was better than that of a single large one. Very few reactions occurred and were not nearly so pronounced as those frequently seen after an intravenous transfusion. Careful citration of the blood will prevent most reactions. There was no evidence of injury to any abdominal organ. There is little possibility of penetrating the bladder or bowel, but care should be taken that they are both reasonably empty. It is virtually impossible to push a needle through either of these organs, unless it is distended. This method should not be used when there is intraabdominal disease or when abdominal discomfort might be a disadvantage, as in severe heart disease or acute pneumonia. It is of little use in the rapid replacement of volume in shock or hæmorrhage, but finds its greatest usefulness in the treatment of secondary anæmias and in combating infection. The authors consider it a safe and effective method and so easy of application as to be available for the use of every practitioner.

Bulbar Poliomyelitis.

JAY I. DURAND (*Journal of the American Medical Association*, October 5, 1929) states that in bulbar poliomyelitis the paralysis of the muscles of respiration, deglutition and phonation generally clears up in from a few days to two weeks. The patient with these forms of paralysis makes a good recovery if he can be carried through the acute stage. The author concludes that death in this type of poliomyelitis occurs frequently from drowning in retained secretions rather than from paralysis. Postural treatment with the head well lowered is a life saving measure.

Erythrœdema: A Deficiency Disease.

M. FINDLAY AND R. STERN (*Archives of Disease in Childhood*, February, 1929) draw attention to the resemblance existing between erythrœdema in children and an apparent deficiency disease in young rats. The latter developed patches of scaly dermatitis and alopecia after two to three weeks of feeding on a diet which was complete except that the sole source of protein was dried crude egg white (deficient in vitamin *E*). The perioral and periocular regions were most affected. After a time the body weight fell, the hind limbs became spastic and the rat assumed a kangaroo-like posture, advancing with a slow ataxic gait. Death was preceded by a rapid loss of weight and by cyanosis and general muscular weakness. Autopsy revealed a complete absence of change in the main organs with a complete absence of fat. The blood manifested a slight leucocytosis. Histologically the muscle fibres of the limbs were very much shrunken; the skin manifested hyperplasia and desquamation; the internal organs were unaffected, but the spinal cord was diffusely infiltrated by small round cells affecting in most instances the whole length of the cord. It was most obvious in the region of the posterior horns into which it extended. Boas whose original work these writers are following, suggested that the drying of the egg white robbed it of some hitherto unknown essential dietetic factor or liberated a toxin. A further test showed that as with the vitamins *B* and *C*, a pregnant adult rat, fed on dried egg white as its sole source of nitrogen, produced young which had signs of a deficiency of vitamin *E*,

that is, with the above syndrome, and which died in twenty-eight to thirty-five days. Adult rats, for example, the mothers in this last experiment, show much more resistance, but eventually develope similar lesions, particularly in the skin. Further, it was impossible to transmit this disease to healthy rats by contact, injection of blood or emulsified cord from the diseased rats. The authors pass on to describe a fatal case of erythrœdema manifesting very close resemblance in the cord findings. Raw liver, raw potato, egg or yeast will all cure erythrœdema in the rat or prevent its onset. Cow's milk and mannite are not so efficient. The authors conclude that the evidence collected favours the hypothesis that erythrœdema is a deficiency disease rather than a toxic development.

ORTHOPÆDIC SURGERY.

Bursitis of the Foot.

PERCY WILLARD ROBERTS (*Journal of Bone and Joint Surgery*, April, 1929) describes the situations of numerous bursæ about the foot and ascribes many symptoms to their inflammation. He points out that many patients are treated with foot supports for symptoms similar to anterior metatarsalgia and they really need rest in bed and other treatment appropriate to the inflamed bursa. He discusses the differential diagnosis and suggests that the correct treatment after conservative treatment has failed, is operative obliteration or removal of the inflamed bursa. Of fifty patients treated by conservative or operative measures all were cured or relieved.

Wrist Dislocations with Associated Nerve Lesions.

R. WATSON JONES (*Proceedings of the Royal Society of Medicine*, June, 1929) discusses various dislocations about the wrist joint and he pays particular attention to forward dislocation of the *os lunatum*. Amongst five thousand fractures and dislocations reviewed there were twelve dislocations of this bone. He holds that excision of the bone should be avoided in most instances. Stern's method of manipulative reduction with Thomas's wrench is very liable to be followed by paresis or paralysis of the median nerve and in his opinion should never be used. Reduction should always be attempted by manipulation which should take a definite order. First, the wrist should be dorsiflexed, secondly, the anterior edge of the dislocated *os lunatum* should be controlled by pressure of the thumb. The wrist should then be slowly flexed so that the head of the *os magnum* is made to engage in the corresponding articular surface of the dislocated bone which slips back into position as flexion is attained. If this desirable event should occur, the wrist is maintained in flexion for ten days and the patient is able to resume full work in six to eight weeks. This manœuvre is likely to be successful only within a week of the injury. If it should fail the wrist joint should be opened by a dorsal incision, the head of the *os magnum* freed of adhesions to the capsule, as these are the cause of difficulty in closed reduction, and then this manœuvre is repeated. The use of raspatories and rougines as levers is deprecated. He claims that most patients so treated achieve a measure of improvement which renders it difficult to distinguish the injured wrist from a sound one.

The Antagonism of Tuberculosis and Infantile Paralysis.

L. LAMY (*The Journal of Bone and Joint Surgery*, July, 1929) states that he has been unable to trace a single instance in which osteo-articular tuberculosis has occurred in a limb affected by infantile paralysis, nor has he found it in the healthy limbs of a patient affected by poliomyelitis. Similarly, visceral tuberculosis is apparently rare and a tuberculous spondylitis with scoliosis and kyphotic spine due to pulmonary tuberculosis is very rare. He raises the question whether this antagonism between the two diseases is due to an active immunity conferred on the patient by poliomyelitic virus.

Acute Osteomyelitis of the Spine.

MYRON O. HENRY (*The Journal of Bone and Joint Surgery*, July, 1929) describes a case of acute osteomyelitis of the spine and emphasizes the difficulty of arriving at a diagnosis in many instances. In the author's patient the deep abscess was drained early and this undoubtedly saved the patient's life and apparently also saved the osteogenetic layers of the periosteum, as new osseous tissue sufficient to prevent deformity or paralysis was formed. The skiagrams revealed a normal spine at first and complete obliteration of the body of the vertebra one month later. Ultimately solid fusion of the adjacent vertebræ without deformity occurred.

Congenital Dislocation of the Hip.

FREMONT A. CHANDLER (*The Journal of Bone and Joint Surgery*, July, 1929) in an anatomical study of bilateral congenital dislocation of the hip occurring in a male infant, concluded that the condition was due to diminution in the amniotic fluid which caused a disproportion between the fœtal ovoid and the uterine cavity. This produced a hyperextension of the lower extremities over the semiflexed arms, resulting in a dislocation of the hips by direct pressure upon the greater trochanters aided by external rotation of the hyperextended legs.

Paralytic Deformities of the Leg.

M. S. HENDERSON (*Proceedings of the Staff Meetings of the Mayo Clinic*, July 10, 1929) summarizes his experiences in reconstructive surgical treatment in paralytic deformities of the leg. He found that transplantation of tendons without correction of balance of the bony structures was generally of no avail. Lateral deviation of the foot caused greater disability than drop-foot. Neurotization of paralyzed muscles was not successful. Operations of which tenodesis was typical, were successful in certain instances. Transference of the insertions of various muscles may be done in suitable circumstances; osteotomy and arthrodesis are also of great value, particularly to stabilize the foot prior to tendon transplantation. In this latter operation it is more satisfactory to sew the tendons directly into bone rather than into another tendon, but it is always necessary to correct deformity as a preliminary procedure. A recognition of the limited field for transference of tendons has led to a gradual tendency to stabilize the foot by arthrodesis. Astragalectomy, as advocated by Whitman, in the author's experience gave better results than any other operation where severe calcaneal deformity had been present, provided the correct technique, including backward displacement of the foot, was attended to. The best results in operations for stabilizing the foot were obtained after arthrodesis and combinations of arthrodesis and transference of tendons, although the Campbell bone-block operation together with triple arthrodesis gave very satisfactory results.

Malunited Fracture of the Neck of the Femur.

HENRY MILCH (*The Journal of Bone and Joint Surgery*, July, 1929) describes a case in which the femur was curved in two planes as a result of a malunited fracture. The simple or cuneiform osteotomy in these circumstances is usually unsatisfactory and better results are obtained by turning up the periosteum, splintering the bone in a longitudinal manner with a broad chisel and mallet, suturing the periosteum, approximating the muscles, closing the skin and applying a plaster spica. The author considers that there is an increased tendency to callus formation and the periosteum itself acts as an internal splint and permits of correction of the deformity after the operation is finished. The shortening which follows the removal of a large wedge, is avoided by this operation.

The Ring of the Thomas Splint.

CHARLES S. YOUNG (*Journal of the American Medical Association*, August 24, 1929) in a study of fitting the ring of the Thomas splint emphasizes the need of a lateral convexity in the medial two-thirds of the medial posterior fourth of the ring and an anterior projection of the medial end of the rigid ring to avoid compression of the adductors. The anterior portion of the ring is not rigid and is formed by a leather thigh cuff. Diagrams illustrate the principles described.

British Medical Association News.

SCIENTIFIC.

A MEETING OF THE NEW SOUTH WALES BRANCH OF THE BRITISH MEDICAL ASSOCIATION IN CONJUNCTION WITH THE VETERINARY ASSOCIATION OF NEW SOUTH WALES was held at the Medical School, University of Sydney, on November 28, 1929, DR. E. M. HUMPHERY, the Vice-President, in the chair.

Tuberculosis in Animals and Man from the Point of View of Public Health.

DR. H. V. D. BARET read a paper entitled: "Bovine Tuberculosis in Man" (see page 100).

MR. J. DRABBLE read a paper entitled: "Tuberculosis and Meat Inspection" (see page 103).

MR. H. R. CARNE read a paper entitled: "Tuberculosis of Animals in Relation to Public Health" (see page 105).

MR. MAX HENRY read a paper entitled: "Bovine Tuberculosis in Man" (see page 108).

DR. F. GUY GRIFFITHS said that the members of the Branch were grateful to the three members of the Veterinary Association for their papers. What struck him most was the difference in the point of view. This arose chiefly because Dr. Baret thought of the effect of bovine tuberculosis on the health of human beings and this was a secondary consideration to Mr. Max Henry. The latter had spoken of herds and of the percentage of herds affected, but although 60% of herds might be affected, there might be no more than one or two tuberculous animals in each herd. It was particularly striking that the three veterinary surgeons accepted a reaction to tuberculin as complete proof of the presence of tuberculosis in the animal. They had the opportunity of examining the reacting animal *post mortem* and of proving that every reaction had a lesion, even though slight.

It was true that if the lesion were slight, it might extend and cause serious disease. Many cattle in New South Wales were milking Shorthorns or Jerseys and these were particularly liable to tuberculosis. It was rare to see Kerry cattle. Dr. Griffiths thought that they owed a great deal to the Veterinary Department for the manner in which they had grappled with the state of affairs and so greatly reduced tuberculosis in New South Wales in the last thirty years. He had been most interested in hearing from Mr. Carne about the tuberculous nature of the dry scaly scabs on parrots; he would examine his own pet parrot next morning. Mr. Carne had not mentioned monkeys. They were of domestic pets the most liable to tuberculosis. In countries with a warmer climate such as the Malay Peninsula and India they were often kept as pets. They might be a danger to human beings; they were infected by man and passed the infection on to other human beings. So, too, might other domestic animals. He was glad that Mr. Carne had emphasized the potential danger of the family cow; it would sometimes be found that the animal which was used exclusively to supply milk to the family was tuberculous. The danger was a very real one. He had been interested to hear the views expressed as to the effect of Pasteurization of milk. He thought that it would be better to aim at supplying milk free of tubercle bacilli. It was only of recent years, since chemical pathology had bulked so largely in their work, that the changes in the iodine, calcium and phosphorus content resulting from Pasteurization had been emphasized. It was sad to learn from Mr. Carne that the effect of *Bacille Calmette-Guérin* in animals was so evanescent and of doubtful value. He was sure that Dr. Baret was right when he stated that bovine tubercle bacilli caused little tuberculosis in human beings except in early life; then it was chiefly in the cervical and abdominal lymphatic glands. Most surgical tuberculosis in children was due to the human type of bacillus, coming from sputum. It was picked up by the finger nails of the baby crawling about the floor. The tuberculous patient was more dangerous than the milk.

He appreciated Mr. Max Henry's remarks about tuberculosis in cattle. It was important that since they had no means of curing diseased cattle, there should be no relaxation in the departmental activities to eradicate it; this would benefit the dairyman and the pastoralist. He thought that it must be very rare for pus in meat to get on to the market. He could reecho Dr. Baret's remarks in regard to infection with bovine tuberculosis. There was no cause for alarm; the incidence was decreasing. The danger came almost solely from milk and less from meat. Rivière went so far as to state that a tuberculous infection in childhood was beneficial to the child. This was, of course, exaggerated, but it was striking to put it in that way. Most children with a tuberculous infection recovered and acquired an immunity which might last through life. Metchnikoff had nursed his wife with tuberculosis and he claimed that his escape from infection was the result of immunity acquired from a mild tuberculous infection in infancy. If Dr. Baret had gone so far as to say that milk was more dangerous on account of the men who handled it, than of the cow who secreted it, he might well agree with him.

MR. J. C. SANDERSON referred to one or two points in Dr. Griffiths's remarks. It was not true that difference in breed in cattle had any influence on tuberculous infection. Every breed in New South Wales was equally affected. In regard to the danger of the private cow, he recalled a friend of his who would not use general milk. He had had three Jersey cows. The friend had asked him to look at the animals. He had palpated the udders and had discovered that two of the three animals were infected by tuberculous mastitis. Two of the members of the friend's family had suffered from tuberculosis. If people desired to keep private cows, they should have the animals tested occasionally. He had been interested in Dr. Griffiths's remarks about the immunity resulting from an infection in childhood. This did not happen in cattle. If a focus was discovered in early life in cattle, the infection later on became an open one and the animal died. This was probably due to the harder life of cattle, the starvation and so forth to which they were subjected. In the hundreds of *post mortem* examinations which they undertook, they had not discovered such an occurrence in an animal.

DR. SINCLAIR GILLIES said that he had been favoured by an advance copy of Dr. Baret's paper. In referring to the difference of opinion that had been evidenced, he said that they got half their fun out of life by discussing statistics and culling them to suit themselves. The one important point arising from Dr. Baret's paper was that his figures showed that the rate of mortality from tuberculosis, thought to be of animal origin, had decreased out of all proportion to that from human source. It suggested that the veterinary surgeons were doing their work better than the medical practitioners. Turning to the preventive side in human beings, he thought that the veterinarians could sit back and that the health authorities would have to put forward greater efforts. Referring to the tuberculin reaction, he said that it must be regarded as a reaction of sensitization. It was a reaction to tuberculous toxin. They did not know how it occurred or how long it lasted. The sensitivity of man to tuberculin might go on through life and the individual might die of some other disease. In regard to animals reacting he wondered whether they were followed through life and whether the veterinary surgeons could say that these did not recover. Reference had been made to the loss of iodine and calcium by Pasteurization. He wanted to know how it was possible to lose iodine. If milk was heated it was possible to lose calcium, but if the milk was allowed to cool in the vessel, much was not lost. He thought that at the present time Pasteurization was of value. As far as the *Bacille Calmette-Guérin* was concerned, he had seen it used extensively in France. The French were very keen on it. The children were either vaccinated hypodermically or in the great majority of cases the bacilli were given by mouth. When a hypodermic injection was given, an abscess formed at the site of injection, the abscess discharged and healing occurred. The child subsequently gave a skin reaction to tuberculin. When the vaccine was given by mouth, the children might go for years without giving the tuberculin reaction. The vaccine evidently did not get further than the intestinal canal. Decreased mortality

in these cases simply meant greater parental care. Dr. Gillies concluded his remarks by saying that he was sorry to see that Dr. Baret had retired from the position of Director of Tuberculosis.

DR. HARVEY SUTTON said that the thanks of the community were due to the veterinarians for the way in which they protected the cattle. He referred to the statement of Dr. Gilruth when the latter had championed the cause of the veterinary surgeon. On seeing a friend who had been told by his medical attendant that he was about to die, Dr. Gilruth had said that if a veterinarian said that one of his patients was going to die, it did die. The veterinarian could kill the beast, he could study the pathological changes and he could thus protect the rest of the herd. The medical practitioner was in a different position in this regard. Dr. Sutton said that he was arriving at the sceptical stage of life and he wondered whether they were not pursuing an illusory ideal in attacking the tubercle bacillus alone. The direct hunt after the bacillus often led them along lines which were not the most valuable. They were often apt to pride themselves on the reduction in tuberculous infection, but Dr. Sutton pointed out that the reduction had been parallel to the increased buying power of money and the increase of wages. In referring to the different positions in the several States in regard to tuberculosis in animals, as shown by the low percentage of human non-pulmonary tuberculosis in Queensland as against the relatively high figures of Victoria and South Australia, he pointed out that there were varying hygienic conditions for the beast rather than control by tuberculin and they had to be careful before they allotted to themselves full credit for any improvement. He believed in Pasteurization under modern conditions of city supply. He did not agree with what had been said about calcium and phosphorus. The calcium and phosphorus in milk was three times in excess of that required by the human being. Ample would remain in Pasteurized milk. Pasteurization stood up to the biological test comparing the growth and health of large numbers of children on milk feeding with Pasteurized milk with that of the ordinary child population. Pasteurization was carried out in order to keep the milk; it was a safeguard that was inevitable in city supplies.

DR. F. S. HONE said that it was difficult not to take sides. Dr. Baret had been accused of special pleading. Dr. Hone thought that all the special pleading was on the other side. It had to be remembered that they could prove anything from statistics. A great deal depended on the point of view. Dr. Baret talked of bovine tuberculosis in human beings and the veterinarians talked of bovine tuberculosis in animals. The late Eustace Ferguson had been unable to find tubercle bacilli in milk after years of search. Only 25% of tuberculosis in childhood was due to bovine tuberculosis. If 75% of the tuberculosis in children and 95% of the tuberculosis in adults was due to the human type of bacillus, greater attention should be paid to the question of the human type of tuberculosis. If half the attention was paid to human tuberculosis that was paid to bovine tuberculosis, they would get better results. Dr. Hone went on to discuss the endowment of research and said that it was possible to get sums of £10,000 for research into tuberculosis in animals, but that the people who were prepared to give the money would have a fit if they were asked to give a similar amount for research into the tuberculosis of human beings. The human problem was undoubtedly the more important. They had no figures to show the extent of bovine tuberculous infection in man. Dr. Holmes, of the Commonwealth Department of Health, was undertaking an investigation to remedy this state of affairs. He was examining specimens obtained from various children's hospitals and was endeavouring to type the bacilli. Dr. Hone concluded his remarks by thanking the members of the Veterinary Association for the part they had taken in the meeting and Dr. Baret for the figures and facts which he had brought forward.

DR. E. M. HUMPHERY expressed the thanks of the members of the Branch to the members of the Veterinary Association for their large share in the discussion of the evening. It was important for medical practitioners to know what was being done from other standpoints. The evening's discussion showed that medical practitioners and veterinarians were dealing with two different diseases. All doctors found that there was a great deal of apathy in human beings. People were careless about conditions of living. As the general public became better educated in regard to the importance of early recognition of infection, there would be more possibility of stamping out the disease.

DR. H. V. BARET said in reply that the main purpose of his paper had been fulfilled. He accepted Mr. Drabble's figures in correction of those given by himself, but did not think that the difference was of serious import. Mr. Drabble had said that the disease was progressive in dairy cows. This was not evidenced by the results of examination of milk or by the results of observations at abattoirs. He denied that his paper could be described as special pleading. New facts had been brought forward and he was glad that Mr. Henry had made them more prominent. He thanked Dr. Griffiths for his remarks. If 50% of herds were involved and there were one hundred animals in each herd, the incidence might be only 0·5%. Dr. Baret referred to samples of milk that had been examined at the Board of Health and said that of 668 samples examined, only one had yielded a positive result. He referred to the work of Chick and to her conclusion that there were so many factors involved that there was no definite evidence against the Pasteurization of milk. He thought that they should continue to use Pasteurization and insist that the source should be as pure as possible.

Mr. J. C. Sanderson, the President of the Veterinary Association, expressed the thanks of the members of his Association for the opportunity afforded for joint discussion. The subject was an important one and it did them all good to look at things from two sides.

A MEETING OF THE SECTION OF PÆDIATRICS OF THE NEW SOUTH WALES BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the Royal Alexandra Hospital for Children on June 29, 1929. The meeting took the form of a series of clinical demonstrations.

Hydrocephalus.

DR. R. B. WADE showed a child, aged ten months, who was suffering from hydrocephalus. At the age of five months the child had suffered from meningococcal meningitis and she had been discharged as cured three months later. At the age of nine months the child had been readmitted with hydrocephalus. The circumference of the head had then measured 46·25 centimetres (eighteen and a half inches). The anterior fontanelle had been five centimetres (two inches) in diameter. The patient had been quite blind. The pupils had reacted sluggishly to light. During the following month the circumference of the head had increased by five centimetres and the anterior fontanelle had been very tense and bulging.

On June 6, 1929, a craniotomy had been performed and a communication established between the ventricles and subarachnoid space by tearing through the velum. Since then the anterior fontanelle had been quite soft and on June 18, 1929, the circumference of the head had become less by five centimetres. The reaction of the pupils had become more brisk and apparently some sight had returned.

Traumatic Meningocele.

Dr. Wade's second patient was a boy, aged fourteen months, who had been admitted on March 8, 1929, after having been kicked by a horse on the back of the head. He had not been rendered unconscious, but had vomited and become drowsy. A large hæmatoma had been present on the occiput. X ray examination of the skull had failed to reveal any abnormality. The hæmatoma had gradually become absorbed. Three weeks later a small pulsating tumour had appeared in the region of the injury. This had been partly reducible and had become tense when the child cried. It had increased rapidly in size within a few days to about 3·75 centimetres in each direction. At the

same time small projections of bone had been felt at the sides. Examination of the fundi had revealed choked discs.

On April 8, 1929, ventricular puncture had been performed and two cubic centimetres of blood-stained fluid had been withdrawn. On puncture of the *cisterna magna* forty cubic centimetres of blood-stained fluid had been removed and the tumour had collapsed completely. The tumour had increased again in size to a slight extent for the next week and had then disappeared. The projecting bone had partly returned to its normal position and the papillitis had disappeared. At the time of demonstration the child had a slight internal strabismus.

Thrombocytopenia.

The third patient shown by Dr. Wade was a boy, aged thirteen years, who had been admitted to hospital on November 4, 1928. According to the history he had suffered from frequent epistaxis which had been treated with calcium lactate and packing. On admission the child had been covered with bruises. Effusions had occurred into the muscles. He had had several attacks since then and for these transfusions had been necessary. Examination of the blood had revealed a secondary anæmia characterized by absence of platelets. After blood transfusion platelets had been present in small numbers. On June 13, 1929, splenectomy had been performed and ten days later the blood platelets had numbered 186,500 per cubic millimetre.

Acholuric Familial Jaundice.

Dr. Wade also showed two brothers, aged eight and six years respectively, who were suffering from acholuric familial jaundice. Since early childhood the boys had suffered from jaundice, intermittent pyrexia lasting a few days, pain in the left hypochondrium associated with the pyrexia, enlargement of the spleen, varying in size, and slight enlargement of the liver. The blood picture had been that of very advanced secondary anæmia with nucleated erythrocytes, polychromasia, anisocytosis, poikilocytosis *et cetera*. The Van den Bergh test had yielded an indirect positive reaction. On testing the fragility of the cells they had been found more resistent to hæmolysis than control cells. On X ray examination associated changes had been found in the skull. Iron, arsenic, extract of liver, raw meat juice *et cetera* had been administered without effect.

On April 18, 1929, splenectomy had been performed on the elder brother. Within a few days almost a dramatic change had occurred in the patient's colour. The jaundiced appearance had vanished and had given place to an ordinary pallor of an uncomplicated secondary anæmia. The findings on blood examination are set out in the accompanying table.

Date.[1]	Erythrocytes.	Leucocytes.	Hæmoglobin Value.	Character of Erythrocytes.
April 10, 1929	1,340,000	5,900	25%	Polychromasia, macrocytes, poikilocytosis, nucleated forms.
April 17, 1929	2,100,000	10,500	35%	Polychromasia, macrocytes, poikilocytosis, nucleated forms.
April 22, 1929	2,290,000	7,700	32%	Pale, no polychromasia, regular, even, no nucleated forms.
April 29, 1929	2,500,000	7,300	49%	Full, regular, even.
May 6, 1929	2,940,000	8,000	47%	Full, regular, even.
May 14, 1929[2]	2,330,000	12,200	50%	Many large forms, fairly full, regular.
May 21, 1929	2,810,000	14,400	48%	Full, regular, even.
May 28, 1929	3,120,000	10,600	56%	Fairly full, regular, even, one normoblast.
June 4, 1929[3]	2,500,000	12,500	50%	Full one normoblast, regular, even.
June 10, 1929	2,000,000	15,200	44%	Full, even, macrocytes present, nucleated forms.
June 18, 1929	2,570,000	15,500	40%	Slightly pale, regular, even.
June 24, 1929	2,320,000	11,800	45%	Full, regular, even.

[1] Blood transfusion, 210 cubic centimetres (7 ounces), was carried out on April 16, 1929, and splenectomy on April 18, 1929.
[2] Change of pathologist.
[3] Change to original pathologist.

Osteomyelitis.

The next patient shown by Dr. Wade was a boy, aged thirteen years, who exemplified the after results of osteomyelitis. The child had suffered from osteomyelitis of the neck of the right femur and of the ramus of the left mandible in August, 1925. As a result there had been complete ankylosis of the left temporo-mandibular joint, causing inability to open the mouth more than 1·5 millimetres, and ankylosis of the right hip joint with 7·5 centimetres (three inches) of shortening and slight adduction deformity.

On May 2, 1929, the left temporo-mandibular joint had been excised and fascia had been inserted between the raw bony surfaces. The mouth had been kept open at night by means of a dental support and this had been removed in the day time. The child had been encouraged to move the jaws by being given unlimited supplies of chewing gum. The result was satisfactory. On June 20, 1929, subtrochanteric osteotomy of the right femur had been performed and the limb abducted about 30° from the middle line. The child was being nursed in a Jones's abduction frame. The relative shortening had been reduced from 7·5 to 2·5 centimetres.

Talipes Equino-Varus.

Dr. Wade also showed a boy, aged six years, who suffered from a relapse of *talipes equino-varus*. He had been treated in early infancy at the Royal Alexandra Hospital for Children and then at the massage department. The parents had ceased bringing the child and his condition had been allowed to relapse. On examination *talipes equino-varus* had been very advanced. Both feet had been rigid and dorsiflexion very poor. The child had been unable to evert the feet. The feet had been rotated inwards from the mid-carpal joint and considerably flattened. *Pes cavus* had been present and the child had walked on the outer edge of both feet with the heels raised from the ground. Treatment had consisted in division of the *tibialis posticus*, the short flexor of the great toe, the inferior calcaneo-scaphoid ligament, the *flexor longus digitorium* and the *tendo Achillis*. The feet had been wrenched into an overcorrected position. The position had been maintained by means of strapping and massage had been given every day. It was intended that the child should wear special boots with the soles raised on the outside.

Imperforate Anus.

Dr. Wade's last patient was a boy, aged eight and a half years, who had been admitted to the Royal Alexandra Hospital for Children at the age of six days with an imperforate anus and passing fæces *per urethram*. Lumbar colostomy had been performed. The child had been readmitted at the age of eight and a half years with a history of frequent attacks of cystitis when pus and what was apparently fæces were discharged from the urethra. The colostomy had functioned perfectly. An operation had been undertaken on June 13, 1929, to cure the fistula and if possible to make an artificial anus further down. The rectum had been found enormously dilated and firmly incorporated with a dilated bladder below the peritoneum. The condition had been quite inoperable and had been that of a persistent urachus.

Syphilitic Ulceration.

DR. P. L. HIPSLEY showed a girl, aged six years, who had been admitted to hospital on June 6, 1929. There was no abnormality in the family history beyond the fact that the first child had been born prematurely at six months and had died at birth. Another had died of pertussis at the age of six weeks. The other two were healthy. The history of the patient's illness was that she had had keratitis at the age of four. This had been cured by mercurial treatment. Fluid had been found in the left knee joint six months later. This had got well after five weeks. The patient had then suffered from pertussis. Three months before admission the patient had suffered from a swelling of the right side of the neck and this had increased. After a month a similar swelling on the left cheek had formed an ulcer. A month before admission

a swelling had occurred on the right side of the neck below the first swelling. On examination of the respiratory system on admission prolonged expiration had been discovered in the left apex anteriorly. The circulatory and nervous systems had been normal. The tonsils had been slightly enlarged and a small ulcer had been found on the right side of the uvula. The cervical glands on the right side of the neck had been enlarged, two swellings had been red and fluctuant and the lower had been just above the clavicle. On the left cheek there had been a dirty ulcer with a punched-out appearance and about five centimetres (two inches) in diameter; its edges had been undermined. On June 21, 1929, the lower of the two swellings had burst and a punched-out ulcer, similar to the other, had been left. No reaction had been obtained to the Wassermann test or to the von Pirquet test and the urine had been normal. The erythrocytes had numbered 4,000,000 and the leucocytes 12,200 per cubic millimetre. The pus had contained pneumococci and staphylococci. No periostitis had been found on X ray examination of the tibiæ and some increased mottling of the lungs had suggested tuberculosis. A provocative dose of 0·2 gramme of "Neokharsivan" had been given on June 25, 1929, and the Wassermann test, carried out twenty-four hours later, had yielded a reaction.

Sclerodermatous Erysipelas.

Dr. Hipsley also showed a boy, aged five and a half months, who had been admitted to hospital on May 31, 1929. There had been a history of pyloric stenosis for which an operation had been performed four months previously. The child had been fed on cow's milk and cream emulsion. Six days before admission the child had drawn up its knees and had become very irritable. The buttocks and legs had become stiff and hard and the knees bent. Four days before admission the legs had become red. There had been no vomiting, but anorexia had been present. On admission the child had been very irritable, the general condition had been good. The respiratory, circulatory and alimentary systems had been clear. Both legs and buttocks had been swollen and red. Red nodular swellings had been most pronounced on the buttock, the posterior portion of the thigh and the anterior aspect of the tibiæ. The legs had been flexed at the knee and the patient had resisted any attempt at extension. The temperature had been practically normal. The patient had taken all food, though unwillingly. On June 4, 1929, both arms had become affected simultaneously. Red nodular swellings had started in the upper part of the arms and had spread towards the wrists till finally the hands had been affected in four days. Two days later the temperature had risen to 38·3° C. (101° F.) and had kept at this level for seven to eight days. The erythrocytes had numbered 3,230,000 and the leucocytes 9,300 per cubic millimetre. The urine had been normal. The patient's condition had been regarded as erysipelas and he had been isolated.

Ichthyol and glycerin had been applied to the swellings. On June 8, 1929, 500,000 units of erysipelas streptococcus antitoxin had been given intramuscularly. Two subsequent doses of antistreptococcal serum had been given. Four days after the first serum injection definite improvement had been noted in all the limbs whose condition had previously remained stationary. The limbs had been less red, less brawny and less painful. Improvement had continued steadily since, the temperature had steadily fallen since and at the time of the meeting had been normal for four days.

Congenital Deformity.

DR. W. VICKERS showed a baby, aged two months, who was suffering from a congenital deformity. The child was a first child. The forearms were contracted on the arms and the forearms were in a position of flexion and internal rotation. Similar deformities existed in the mother, grandmother, an aunt and two uncles.

Posterior Capsuloplasty.

Dr. Vickers also showed a girl, aged six years, who had suffered from what was regarded as Still's disease four years previously in Scotland. The patient had been admitted to the Royal Alexandra Hospital for Children in March, 1928, with practically all the joints involved, including the spine. Most of the joints had been contracted. Ultimately subluxation of the tibiæ on the femora had occurred and the operation of posterior capsuloplasty had been performed on May 14, 1929. The limb had been put up in plaster with the knee in extension. Plaster had been removed one week later and a little flexion at the knee had occurred. The limb had been straightened under anæsthesia on June 7, 1929, and again put in plaster, reinforced posteriorly by aluminium.

Aortic Regurgitation.

DR. M. J. PLOMLEY showed a boy, aged seven and a half years, who had been admitted to hospital on June 11, 1929. Ever since the child was six months old the parents had known that he had heart trouble. It had not affected his health. The child had been breathless on exertion ever since an attack of pneumonia six months previously. He had lost weight and complained of pain in the left arm and leg. On examination there was no sign of cardiac failure. The pulse was collapsing in type. The systolic blood pressure was 105 and the diastolic pressure 40 millimetres of mercury. The apex beat was in the sixth intercostal space ten centimetres (four inches) from the middle line. There was a palpable systolic thrill over the lower end of the sternum and in the epigastrium. Scattered rhonchi were audible in the lungs. X ray examination of the heart revealed an increased shadow and the shape suggested hypertrophy rather than dilatation.

Anæmia.

Dr. Plomley's second patient was a boy, aged four years, who had been admitted to hospital on April 9, 1929. He had lived on fluids ever since birth and had seemed to choke when he was given solids or semi-solids. Five months before admission he had begun to refuse fluids and five weeks before admission he had lost power in the legs and had become pale. The family history was clear; there were ten healthy other children. The child was pale and poorly nourished and poorly developed. The muscles were extremely flabby. It could not talk or betray any emotion other than *ennui*. Small discrete and shotty glands were palpable in the neck and groins and on examination of the abdomen no enlargement of the spleen or liver was palpable. The other systems were normal. No reaction had followed the von Pirquet test. On April 11, 1929, the erythrocytes had numbered 2,800,000 and the leucocytes 5,800 per cubic millimetre. The hæmoglobin value had been 32%. No reaction was obtained to the Wassermann test. On X ray examination enlarged glands were seen in each hilar region. On June 11, 1929, the erythrocytes had numbered 2,900,000 and the leucocytes 2,750 per cubic millimetre. The hæmoglobin value had been 40%. The fragility of the erythrocytes had been less than normal.

Hodgkin's Disease.

Dr. Plomley's last patient was a boy, aged two years and seven months, who had been admitted to hospital on May 29, 1929, with the history that lumps in the neck, groins and armpits had been present for six weeks, that pallor had been present for this time and that the abdomen was enlarged. Examination had revealed a well developed child with a definite degree of anæmia, enlargement of the liver and spleen and of the cervical and lymphatic glands. Three blood counts had been made between May 31 and June 25, 1929. The erythrocytes had varied between 2,970,000 and 2,340,000 per cubic millimetre and the leucocytes between 7,000 and 2,500. The hæmoglobin value had varied between 32% and 43%, the colour index between 0·19 and 0·85; the polymorphonuclear leucocytes had risen from 2% to 51% and the lymphocytes had fallen from 85% to 38%. No reaction had been obtained to either the Wassermann or the von Pirquet test. After the patient's admission he had suddenly become more pale and examination had revealed destruction of erythrocytes. Liver extract had been given and considerable improvement had resulted.

Splenic Anæmia with Hæmatemesis.

DR. MARGARET HARPER showed a girl, aged twelve years, who had been admitted to hospital on January 1, 1928, with a history of having been ill for five days. The illness had commenced with vomiting and on the following day the patient had vomited 300 cubic centimetres of blood. No pain had been present, but some melæna had occurred. On examination the child had appeared well nourished, but had been very pale. The spleen had been 6·25 centimetres (two and a half inches) below the costal margin. There had been no tenderness. Two days after admission the patient had vomited large quantities of bright red blood with clots and two hours later had vomited more blood. The spleen had definitely decreased in size after this hæmorrhage. Blood transfusion of 300 cubic centimetres of blood (ten fluid ounces) had been given and three days later another transfusion had been carried out. The patient's general condition had not been so good and the spleen had begun to increase in size. Another transfusion of 240 cubic centimetres had been given with good result. This had been repeated three weeks later. After this liver extract and arsenic had been given and definite improvement had resulted. At the time of the meeting the results of liver efficiency tests were normal. Dr. Harper showed in tabular form the results of the examination of the blood and pointed out that the number of erythrocytes had increased from 1,400,000 to 3,360,000 per cubic millimetre. The hæmoglobin value had improved from 25% to 60%. The leucocytes at first had numbered 9,200 per cubic millimetre, but at the time of the meeting they numbered 3,300.

Myeloid Leuchæmia.

DR. C. L. S. MACINTOSH showed a girl, aged twelve months, who had been admitted to hospital on February 1, 1929. The child was a "Lactogen" baby. There was no family history of tuberculosis. Two weeks prior to admission the child had begun to vomit. It had continued to take its food well, but had vomited after taking food. There had been no diarrhœa and no hæmorrhage. Fever had occurred. At the time of admission the vomiting had ceased. On examination the child had appeared pale, but well nourished, fretful, but not looking very ill. A large swelling, evidently the moderately enlarged spleen, had been present in the left flank. The liver had been enlarged. The cervical glands had been palpable. It was pointed out that since admission the child had gone down hill. For a time there had been an increase in weight, but a fairly rapid loss was taking place. The child was extremely pale and looked very ill. The spleen had definitely increased in size and was well beyond the middle line. The spleen varied in size. Abdominal distension had always been very pronounced. A profuse nasal discharge had been present, but when it was submitted for examination, no Klebs-Löffler bacilli had been found. Of late the child had been in the habit of lying on her "spleen." There had been variations in the blood counts as follows: The erythrocytes had varied between 3,200,000 and 1,550,000 and the leucocytes between 222,000 and 190,000 per cubic millimetre. The colour index had varied between 1·07 and 0·66, the hæmoglobin value between 50% and 20%, the polymorphonuclear cells between 69% and 37% and the combined lymphocytes between 25% and 43%.

Neuroblastoma.

Dr. Macintosh also showed a patient who was regarded as suffering from neuroblastoma. The patient was a girl, aged nineteen months, who had been admitted to hospital on May 19, 1929. The family history contained nothing of note and there was no history of tuberculosis. The patient's health had been good. Six weeks prior to admission the child had suffered from a "cold in the eyes" with fever, but no vomiting. A week later the child had been unable to use the right leg, but had soon appeared to improve. During the week prior to admission the left leg and right arm had appeared weaker and the right shoulder had not been normal. The child had appeared to be tender to the touch. It had pulled at both its ears and had been becoming paler. On examination the child had been pale and irritable, but could sit comfortably in bed. There had been no evidence of paralysis, but apparently some muscle wasting had occurred. When any part of the child had been touched, pain had apparently resulted and the child had cried when it was approached. Since then a good deal of this tenderness had been lost. The child lay without attempting to sit up, but was able to sit up comfortably when placed in a sitting position. The Wassermann test had been carried out on two occasions, but no reaction had been obtained. This test had also been applied to both parents without result. The erythrocytes had increased from 2,670,000 to 3,750,000. The hæmoglobin value had remained at 40%, but the colour index had dropped from 0·7 to 0·53. The erythrocytes were pale and even, while some were larger and some were smaller than normal. The white cells had increased from 7,800 to 17,200 per cubic millimetre and the percentage of polymorphonuclear cells had increased from 18 to 55. The lymphocytes had diminished from 67% to 38%. Soon after admission 6% of "young" lymphocytes had been present. On X ray examination advanced periostitis of the long bones had been discovered. There were also present large areas of rarefaction, circular and circumscribed.

Retroperitoneal Hæmorrhage and Hæmophilia.

Dr. Macintosh also showed a boy, aged nine years, who had been admitted to hospital on June 21, 1929. The family history contained nothing of note. There was no history of hæmophilia. The child had suffered from hæmophilia from the time when he was a few months old, but he appeared to be "growing out of the trouble." In one year he had been admitted to hospital on nineteen occasions on account of hæmorrhage. Hæmorrhage had occurred in practically every part of the body, subcutaneous tissue, conjunctiva, from the nasal mucous membrane, in the sublingual tissues (when the swelling had been too great to permit of feeding other than by the nose), from tooth sockets, from cuts as well as into various joints and into the peritoneal cavity. Previous treatment had included the administration of calcium salts, serum, parathyreoid gland extract and various hæmostatic substances as well as transfusion of the father's blood. As far as could be remembered the hæmorrhages had nearly always been multiple and had apparently been arrested by absorption of the patient's own blood and after each attack the period of freedom from hæmorrhage had gradually increased. The attack for which he had been admitted, had been caused by a fall four days before admission when he had caused his nose to bleed. Two days later he had experienced abdominal pain in the right side together with incessant vomiting. He had become paler. On examination on admission there had been a large mass in the right flank; this had been very tender and dull on percussion. The abdominal muscles had been rigid, especially on the right side. The muscles had not moved with respiration. The mucous membranes had been blanched and the child had looked very ill. An immediate transfusion of his uncle's blood had been carried out. On June 25, 1929, a hæmatoma of the right groin had been noticed. At this time the coagulation time had been forty-six and a half minutes.

Ascites.

The fourth patient shown by Dr. Macintosh was a boy, aged eight years, who had been admitted to hospital on May 28, 1929, suffering from ascites of uncertain origin. According to the family history the mother had had two premature children during the seventh and eighth month of pregnancy respectively; these children had died when one and six days old. The patient had also been born prematurely and had not thriven during the first six months of life. He had not been well since an attack of measles six months before admission. On examination he had appeared comfortable, but very wasted. The abdomen had been moderately distended and had contained free fluid. The superficial veins had been prominent. The liver had not been palpable and the area of its dulness had been diminished. The spleen had not been palpable nor had a mass of any description been felt. There had been no tenderness. The distension had increased and paracentesis had been necessary on three occasions. Both the von Pirquet and the Wassermann test had been carried out twice and no reaction had been obtained to either.

The radiologist had suggested the presence of a left basal pleurisy. On June 17, 1929, 720 cubic centimetres (twenty-four fluid ounces) of ascitic fluid had been removed. This had been blood stained and on microscopical examination had been found to contain erythrocytes, leucocytes and occasional endothelial cells. No organisms had been discovered and no growth had followed an attempt at culture.

Cretinism.

DR. WILFRED EVANS showed a girl, aged eight years, a cretin, who had been admitted on May 9, 1929. The patient had contracted measles while in hospital. Dr. Evans drew attention to the fact that the *sella turcica*, the skull and the mandible were normal on X ray examination and that rhachitic changes which were chiefly old, were present in the knee. Both patellæ were ill developed and ossification was delayed. The patient had manifested symptoms of megalocolon and on X ray examination the sigmoid had been found to be extremely redundant and the radiographer had reported that no definite Hirschsprung's disease was present. The patient's blood serum calcium was 11·8 milligrammes per hundred cubic centimetres. Thyreoid extract in doses of 0·015 gramme (one quarter of a grain) had been given twice a day and the patient had been discharged. On readmission on June 25, 1929, the patient's weight had been 25·7 kilograms (four stone one pound); the normal weight for such a child was 23·4 kilograms (three stone ten pounds). The patient's height was 107·5 centimetres (three feet seven inches); the normal height for a child of the same age was 116·25 centimetres (three feet ten and a half inches).

Oxycephaly.

DR. L. DEY showed a child who was suffering from oxycephaly, webbed hands and feet and a cleft palate. The family history was clear. The child had been born with an ill-shaped head and with bulging eyes. The child was fairly well nourished, the head was elongated and dome-shaped, the forehead was curved and the face receding. The eyes bulged forward on to the cheeks and ptosis of both eyelids was present. The anterior and posterior fontanelles were widely open and a complete tract of membrane could be felt from the nasion to the inion. The child had considerable nasal obstruction and recession of the chest was present. On X ray examination the frontal and parietal bones had appeared to overlap and it seemed that atelectasis of the right lung was present. There was some displacement of the heart to the right. On examination of the *fundi oculorum* no evidence of optic neuritis had been discovered.

NOMINATIONS AND ELECTIONS.

THE undermentioned have been nominated for election as members of the New South Wales Branch of the British Medical Association:

Kelly, John Augustine, M.B., B.S., 1927 (Univ. Sydney), c.o. Dr. R. H. Macdonald, South Grafton.

McEwen, Ronald James Grohan, M.B., Ch.M., 1926 (Univ. Sydney), 284, Clovelly Road, Clovelly.

Old, George Greensill, M.B., Ch.M., 1900 (Univ. Sydney), Stockinbingal.

Australasian Medical Congress (British Medical Association).

PATHOLOGICAL MUSEUM.

IN THE MEDICAL JOURNAL OF AUSTRALIA of October 19, 1929, a short paragraph was published on the museum of the third session of the Australasian Medical Congress (British Medical Association), Sydney, 1929, in which it was announced that some details of the exhibits would be announced in a later issue.

DR. KEITH INGLIS presented a series of specimens illustrating neoplastic disease of bone. These included osteogenic sarcoma, simple bone cysts, central giant cell tumours and some metastatic growths in other organs. He also displayed specimens from the War Pathological Collection, including gun shot wounds of limbs and various organs, concussion effects in such organs as the spleen and kidney, injuries to the calvarium, specimens demonstrating the effects caused by bacillary dysentery, amœbic dysentery and bilharzial dysentery and other tropical affections.

DR. LESLIE UTZ exhibited specimens of metastatic carcinomatous deposits in the spleen, brain, lungs and liver from a primary tumour of the testis. The skin changes of the same patient who had had ichthyosis were also demonstrated. His second series of specimens was of chorion epithelioma of the lungs. Both primary and secondary growths were shown. The third series comprised specimens of hypernephroma. The last series consisisted of specimens from a patient who had died of a pituitary tumour with metastatic growths in the testicle.

DR. REGINALD BRIDGE exhibited eight specimens illustrating the various phases of renal tuberculosis. In the early stages only a few pyramids were involved; in the more advanced there was extensive caseation with the characteristic loculi formation. One kidney was a mere shell; the tuberculous lesion was of many years' standing. There was also a specimen of a guinea-pig to illustrate the characteristic tuberculomata of the peritoneum after tuberculous urine had been injected into the peritoneal cavity.

DR. M. S. S. EARLAM placed on view a series of twenty-seven renal neoplasms from the Department of Urology of the Royal Prince Alfred Hospital. The series was made up of nineteen specimens starting with mixed tumours of the Wilms type, passing on to spindle-celled sarcoma, simple epithelial tumours, papillary cystadenomata, cystomata and other forms of malignant epithelial tumours of the Grawitz type. A second series comprised five neoplasms of the renal pelvis. They were all epithelial growths. They were a villous papilloma, a villous carcinoma, an infiltrating carcinoma and a carcinoma complicating calculus formation. The third group of specimens comprised three tumours gained in the *post mortem* room. The first was a lympho-sarcoma spreading from the retroperitoneal lymphatic glands to the kidney; the second and third were metastatic squamous epitheliomata of the kidney, the primary growths being in the *cervix uteri* and the lung.

DR. A. BOLLIGER and DR. M. S. S. EARLAM displayed a well ordered series of sixteen gross specimens and eleven photomicrographs to illustrate their experimental work in the production of renal disease in dogs by X radiation. The five stages of changes induced by the effects of X rays were illustrated in ten specimens; in the remaining specimens the normal kidney was removed and the second organ was exposed to the action of X rays. When the damage inflicted was severe, the organ was incapable of supporting life. The extreme grades of changes were therefore not seen in this series. One kidney had undergone almost complete repair.

DR. A. H. TEBBUTT showed a series of pathological specimens, drawings and photographs. In one group there were specimens of livers affected by syphilitic hepatitis; both cirrhotic and gummatous lesions were seen in one liver. The remaining specimens were thyreoid glands with simple tumours, malignant tumours and goitrous lesions. The simple tumours included one group of large adenomata which had given rise to purely local signs and symptoms. In some organs the tumours had replaced a lobe, traces being found at the poles or stretched over the surface of the tumour. In the second group there were large or multiple adenomata associated with hyperthyreoidism, the so-called hyperfunctioning or toxic adenomata. The clinical histories of the patients from whom the glands had been removed, were attached. In the third group were large or multiple adenomata associated with hypothyreoidism. Two specimens of malignant tumours, both carcinomata, were exhibited. Diffuse and adenomatous goitres, both associated and not associated with signs of hyperthyreoidism, were also displayed. It was stated that these

goitres were uncommon in Sydney, but were encountered from time to time. A number of diffuse hyperplastic goitres and exophthalmic goitres was shown. Some were diffuse or non-nodular, while in other specimens small adenomata were detected on careful examination. Two specimens of lymphadenoid goitres were shown. It was claimed that this form was apparently commoner than Riedel's struma which was held to represent the later and completed lesion. Each specimen was accompanied by microscopical and clinical notes and records of the after histories of the patients. It was announced that this series of specimens would be displayed permanently in the museum of the Royal Prince Alfred Hospital.

DR. H. R. G. POATE exhibited a series of pathological specimens to illustrate his paper on the so-called benign adenomata. The organs were the mammary gland and the salivary glands.

DR. G. F. F. DAVIES had an exhibit of pathological specimens. Included was one of an embolism of the pulmonary artery. The embolus had been formed of a long blood clot, partly red with patches of white thrombus. It had evidently formed in a small vein. There were also several ovarian tumours, including carcinomata, carcinoma-sarcoma and dermoid cysts containing well developed teeth. There were two specimens of carcinoma of the stomach supervening on chronic ulcers. Macroscopically there was little to suggest that malignant change had taken place. A specimen of hæmorrhagic broncho-pneumonia of influenzal origin was shown. The patient, a female of sixteen years, had died of influenza in 1929. The pathological changes were similar to those seen in the earlier epidemics. The last specimens were kidneys from patients who had suffered from chronic nephritis.

DR. T. CHERRY placed on view microscopical specimens of tumours found in mice that had been inoculated beneath the cuticle with very small doses of virulent tubercle bacilli. The tumours were a papilloma of the urinary bladder, carcinoma of the large intestine, of the stomach, of the pylorus, of the pancreas, of the axillary mamma, of the inguinal mamma and of the hair follicles, sarcoma of the skin and of the head of the humerus, chondro-sarcoma of the lumbar vertebra, generalized sarcoma of the abdomen and angio-sarcoma of the uterus.

DR. HEATHER ROSS exhibited seven specimens of eyes, including infective cyclitis after injury, retinal glioma, tuberculosis of the ciliary body, retinal detachment in a child of eleven years, probably due to a neoplasm, acute glaucoma caused by a swollen cortex in a miniature cataract with obliterated anterior chamber, flat sarcoma of the chorioid, melanotic sarcoma of the ciliary body. She also showed an appendix and caecum with extensive tuberculous infiltration, removed at laparotomy from a woman aged sixty-one, who had symptoms of partial intestinal obstruction. Her last specimen was a liver with multiple abscesses complicating a suppurative pylephlebitis.

The Department of Anatomy of the University of Sydney displayed an exhibit consisting of the following items:

A dissection of the vertebral sympathetic ganglionic chain by Dr. Potts and also some dissections of the brain by the same worker; a dissection to reveal the relations of the vein of Galen or *vena cerebri magna* to the splenium of the *corpus callosum* to illustrate Stopford's suggested explanation of certain forms of papillœdema, prepared by Mr. Harris, prosector for Dr. A. W. Campbell; a series of casts and dissections of the female pelvis by Dr. F. A. Maguire; dissections to illustrate the surgical anatomy of the abdominal walls, by Dr. B. Fraser; casts of aboriginal heads; two specimens to display the anatomy of the middle ear and one dissection of the inguinal region prepared by Mr. Sofer Schreiber and Mr. Rosen, fourth year students of medicine.

DR. LOCKYER POTTER'S exhibit comprised several dissections to illustrate a series of operations designed to overcome the characteristic residual deformity of the hand following suture of the ulnar nerve. The operations themselves constituted the application of the principle of stability, an essential in the preservation of effective movement. The various stages of the operations were illustrated by enlarged photographs with explanatory legends. Some casts of hands displayed the effect of operations for ulnar paralysis with varying degrees of deformity, accentuated by secondary contractures. He showed some splints made of aluminium and others made of celloidin and stockinet. Some of these were exhibited on casts of hands. They illustrated the doctrine of rest to injured parts, applied in the treatment of ulnar paralysis. Four portfolios of photographic studies of war injuries were included. These were concerned with deformities and disabilities of the upper and lower extremities after nerve injury, limping, "poker" back and so on. The functional results following tendon transplantation were also illustrated.

DR. PERCIVAL PICKERILL exhibited a series of wax casts and photographs to illustrate various pathological and traumatic lesions and their restoration by plastic operations. The lesions included malignant growths of the lips, face, forehead and hand and traumatic defects of the same regions. A series of transparencies was also shown illustrating extensive lupus and syphilitic losses and the repair effected by plastic means. A further series of transparencies depicted the restoration of function after facial paralysis by grafts of the temporal and masseter muscles into the *orbicularis oculi* and *orbicularis oris*. A large wax model was shown to illustrate the incisions, flaps and grafts used in the facial restoration.

The members of the Faculty of Veterinary Science of the University of Sydney placed on view specimens, cultures and other exhibits to illustrate actinomycosis of domestic animals. The organisms involved are *Cohnistreptothrix israeli*, *Nocardia bovis*, actino bacillus and staphylococci.

Several exhibits were submitted by members of the Faculty of Agriculture. The part played by bacteria in agricultural practice was illustrated by specimens of root nodules and the organisms found in them, by cultures of nitrogen fixing bacteria and other types of beneficial bacteria. Some of the recent advances in plant pathology were illustrated including the results of investigations into flag smut of wheat and rust of cereals. The specialization of the fungi of rust of cereals was displayed. Various results obtained in the breeding of disease resisting types of wheat were illustrated with living material and compared with established Mendelian phenomena. Other specimens of typical bacterial, fungal and physiological diseases of plants were included.

A comprehensive exhibit was prepared by the Poison Plants Committee of the Council of Scientific and Industrial Research. Dr. H. Finnemore showed the methods of extracting poison plants and of isolating and dealing with their active principles. He also exhibited an improved Fowler's solution compatible with metallic and alkaloidal salts.

DR. R. H. SEDDON exhibited specimens of plants investigated for their poisonous properties to cattle and sheep and preserved tissue of animals poisoned by various poisonous plants. The toxic plants included *Marsdenia rostrata*, *Solanum sturtianum*, *Pimelea pauciflora*, *Acacia glaucescens*, *Euphorbia Drummondii*, *Macrozamia spiralis*, *Euphorbia peplus*, *Eremophila maculata*, *Stachys arvensis*, *Cheilanthes tenuifolia*, *Echinopogon ovatus*, *Lantana crocea*, *Pteridium aquilinum*. Further observations are required in connexion with *Hypericum perforatum*, *Medicago denticulata*, *Cucumis myriocarpus*, *Hypochaeris radicata*, *Euphorbia lathyrus*. The following were found to be non-toxic: *Euphorbia Eremophila*, *Inula graveolens*, *Lantana camara*, *Solanum triflorum*, *Argemone mexicana*, *Boussingaultia baselloides*.

The exhibit also contained preparations from the Australasian Pharmaceutical Formulary.

DR. E. SYDNEY MORRIS planned an exhibit from the Department of Public Health of New South Wales. The individual exhibits included a garbage incinerator of approved design, a piggery constructed for the purpose of keeping pigs in a clean and healthy condition, a rain

water separator for excluding the first rain with the washings from the roof from the storage tanks, a model of an insanitary building, a model of the ward at Waterfall for patients suffering from tuberculosis, a châlet for tuberculous patients, fly-proof windows, a fly trap, a manure bin and a mosquito proof tank strainer. There were also a section of a water pipe lined with cement, some drain pipe junctions, grease traps, a model of a drainage system from a dwelling to the sewer, chemical closets, septic tanks and samples of tank effluent.

DR. A. M. DAVIDSON displayed some objects of interest made by medical practitioners. They comprised a grandfather clock case of Queensland maple, a drug cabinet of silky oak with glass shelves and enamelled white interior, a lady's handkerchief box of rosewood with carved lid, the carving being a copy of a carving of the Jacobean period representing the pomegranate conventionalized and a fire-screen of rosewood carved in a conventional vine pattern.

Public Health.

NOTIFICATION OF PULMONARY TUBERCULOSIS IN NEW SOUTH WALES.

THE Director-General of Public Health is directing the attention of medical practitioners in New South Wales to a proclamation dated March 1, 1929, requiring medical practitioners in all parts of the State of New South Wales to notify pulmonary tuberculosis. Previously this disease was notifiable in part of the State. According to the *Public Health Amendment Act*, 1915, if a medical practitioner "becomes aware" that a person is suffering from pulmonary tuberculosis, he must ascertain whether the sputum contains tubercle bacilli and if bacilli are found, he must notify the authority forthwith on the prescribed form.

Correspondence.

TONSILLECTOMY.

SIR: I cannot seriously believe that your correspondent Dr. R. W. Hornabrook is ignorant of the function of the uvula. I take it that he asks this question in order to introduce an attack upon the modern operation of tonsillectomy by dissection. To quote his own words: "In the big majority of cases the tonsils can be completely and effectively removed by the simple blunt guillotine method in the hands of an operator who knows his work."

Yes; but it is the admitted minority of cases in which the tonsils cannot be completely and effectively removed by the blunt guillotine, that demands a more effective operation which I am sure that surgeons who are competent to speak on this subject, agree is satisfactorily met by the dissection method.

I do not agree with Dr. Hornabrook that dissection is "slow torture"; in most cases it is not slow and it could never be described as torture, that is with a competent anæsthetist.

I most emphatically disagree with him when he speaks of the increased risk of post-operative hæmorrhage following the dissection operation associated with continuous anæsthesia. One of the most important advantages of the dissection operation performed in conjunction with continuous anæsthesia by the Shipway or allied technique, is the facility the operator has of making sure that all the tonsil has been removed and that all bleeding has ceased when the patient leaves the operating table. This cannot be done by the rushed guillotine method (combined with a rapid and sanguinous scraping of the post-nasal region) whilst the patient is coming out of the anæsthetic.

I feel that Dr. Hornabrook's letter is misleading to the profession as a whole and the public who read this journal, and I am confident that the majority of surgeons will support me when I say that the modern dissection operation of tonsillectomy is the only one in which in all cases the tonsils can be completely, effectively and safely removed.

Yours, etc.,

ESMOND F. WEST.

Adelaide.
December 8, 1929.

SIR: Dr. Hornabrook and "Practitioner" should be applauded rather than sneered at for the outspoken manner in which they have condemned the method of enucleation of tonsils by dissection which is being carried out in such a wholesale fashion by many specialists today.

In the same issue of the journal appears a letter by Charles F. Warren, of the Sydney Hospital. Why the necessity for him to be so ungenerous and unjust in his criticisms?

A year or so ago, at a meeting of the Gippsland Division of the Victorian Branch of the British Medical Association, I stated that in my opinion the operation of tonsillectomy by dissection would come into disfavour and that the pendulum would eventually swing back again to the guillotine.

It is pleasing to see that there is evidence of the swing having already begun and that at last the profession is waking up to the fact that this method of operation, quite apart from its resulting long convalescence and extra expense to the patient, is followed by far too high a percentage of fatal and nearly fatal results from post-operative hæmorrhage.

With a blunt guillotine, preferably one that has a serrated edge, and a quick turn of the wrist practically the whole of the tonsil can be removed from its bed and with no hæmorrhage.

This method is applicable to nearly all tonsils with perhaps a few exceptions which it seems a pity require operating on at all.

Yours, etc.,

G. ALF. HAGENAUER.

Sale.
December 17, 1929.

SIR: The letters in your columns of Dr. Hornabrook and "Practitioner" *re* tonsillectomy need further answer in addition to that of my esteemed senior and tutor, Dr. Warren. "Practitioner" allows his ignorance to lead him into insult in his suggestion that some of the modern tonsil experts examine their conscience; rather would I suggest that he examine himself and find a comparison with those who in their self-willed ignorance imprisoned and tortured the scientists of old who dared to express the result of their investigations.

Has "Practitioner" ever seen the mouth and fauces full of blood while the second tonsil is removed per guillotine? If not, I fear his experience is sadly limited. Compare this with his "awesome instruments" which, if he takes the trouble to look, are designed to give airway, vision and a freedom from obstructive blood and mucus.

Has "Practitioner" bestirred himself to compare the death statistics of dissection and guillotine operations, the latter an easy winner?

For my part a lengthy experience of both methods has left me impressed as follows: Many who, contented in their skill with the guillotine, believe they remove the tonsils, fail to do so. It has fallen to my lot on many occasions to control for others the after hæmorrhage of a guillotine operation, even in children, and I know some who still believe the operation a perfectly safe one in spite of such happenings.

Why this constant controversy *re* tonsillectomy? The modern dissection operation is safe and as near to perfect as one could desire. The intratracheal anæsthetic, even with your correspondent's belittled machinery, does enable the surgeon to know that the tonsil is out without deceiving

himself. It is safe and it does enable hæmorrhage to be definitely stopped without the patient being first nearly suffocated with blood and mucus or perhaps plugged up for a long time with pressure swabs, while the doctor hopes that Nature will be kind to him and leave a dry fossa when the plug is removed.

May be it is "Practitioner" himself who has been really unfair to his patients and should examine his conscience. I would advise that he really endeavour to become skilled at dissection himself, persuade his neighbour to learn the use of an intratracheal machine and let the sisters in one hospital become skilled in the use of the instruments; after a conscientious trial over a period of years his opinion as to the merits of the two methods in adults would then be of value to those reading your columns.

Yours, etc.,

DOUGLAS G. CARRUTHERS, M.B., Ch.M.,
Honorary Assistant Ear, Nose and Throat Surgeon, Sydney Hospital; Honorary Consultant Ear, Nose and Throat Surgeon, Canterbury Hospital.

Sydney.
December, 1929.

Books Received.

NEUROLOGICAL EXAMINATION, by Charles A. McKendree, A.B., M.D., with a Foreword by H. A. Riley, A.M., M.D.; 1928. Philadelphia: W. B. Saunders Company; Melbourne: James Little. Post 8vo., pp. 280, with illustrations. Price: 16s. 6d. net.

HOOKWORM DISEASE: ITS DISTRIBUTION, BIOLOGY, EPIDEMIOLOGY, PATHOLOGY, DIAGNOSIS, TREATMENT AND CONTROL, by A. C. Chandler, M.Sc., Ph.D.; 1929. London: MacMillan and Company, Limited. Royal 8vo., pp. 500, with illustrations. Price: 21s. net.

TESTICULAR GRAFTING FROM APE TO MAN, by Serge Voronoff and George Alexandrescu; Translated by Theodore C. Merrill, M.D.; 1929. London: Brentano's, Limited. Demy 8vo., pp. 133, with illustrations.

ULTRA-VIOLET RAYS IN THE TREATMENT AND CURE OF DISEASE, by Percy Hall, M.R.C.S. (England), L.R.C.P. (London), with an introduction by Sir Henry Gauvain, M.A., M.D., M.C. (Cambridge), F.R.C.S., and Leonard E. Hill, M.B. (London), F.R.S.; Fourth Edition; 1929. London: William Heinemann (Medical Books), Limited. Demy 8vo., pp. 266, with illustrations. Price: 12s. 6d. net.

Diary for the Month.

JAN. 22.—Victorian Branch, B.M.A.: Council.
JAN. 25.—Queensland Branch, B.M.A.: Council.
JAN. 28.—New South Wales Branch, B.M.A.: Medical Politics Committee.

Medical Appointments.

Dr. William Bruce Kirkland (B.M.A.) has been appointed Chief Protector of Aboriginals for Central Australia.

Medical Appointments Vacant, etc.

FOR announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser," page xx

BUNDABERG HOSPITALS BOARD, QUEENSLAND: Junior Resident Medical Officer.

RENWICK HOSPITAL FOR INFANTS, SUMMER HILL, SYDNEY, NEW SOUTH WALES: Resident Medical Officer.

WANGARATTA DISTRICT HOSPITAL: Resident Medical Officer.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 21, Elizabeth Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company, Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Members accepting apopintments as medical officers of country hospitals in Queensland are advised to submit a copy of their agreement to the Council before signing. Brisbane United Friendly Society Institute. Mount Isa Hospital.
SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide.	All Lodge Appointments in South Australia. All Contract Practice Appointments in South Australia. Booleroo Centre Medical Club.
WESTERN AUSTRALIAN: Honorary Secretary, 65, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (Wellington Division): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Medical practitioners are requested not to apply for appointments to positions at the Hobart General Hospital, Tasmania, without first having communicated with the Editor of THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to "The Editor," THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, Sydney. (Telephones: MW 2651-2.)

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